C81
1924/25
cop. 2

Legislative Document (1926) No. 29

STATE OF NEW YORK

REPORT

OF THE

New York State Veterinary College

At Cornell University

For the Year 1924-1925

THE LIBRARY OF THE
MAR 30 1926
UNIVERSITY OF ILLINOIS

ALBANY
J. B. LYON COMPANY, PRINTERS
1926

Legislative Document (1926) No. 29

STATE OF NEW YORK

REPORT

OF THE

New York State Veterinary College

At Cornell University

For the Year 1924-1925

THE LIBRARY OF THE
MAR 30 1926
UNIVERSITY OF ILLINOIS

ALBANY
J. B. LYON COMPANY, PRINTERS
1926

C81
1924/25
cop. 2

CORNELL UNIVERSITY

ITHACA, NEW YORK

OFFICE OF THE PRESIDENT

August 3, 1925

The Honorable ALFRED E. SMITH, *Governor of the State of New York, Albany, N. Y.:*

SIR.—I submit herewith a report of the New York State Veterinary College at Cornell University for the year ending June 30, 1925, in accordance with the requirements of the act of the Legislature, approved May 22, 1897 (Chapter 689 of the Laws of 1897) providing for the administration of this college by Cornell University.

The report contains a detailed account of the work the college is doing for the benefit of the veterinary profession and the live stock owners of the State. It gives the results of researches on animal diseases that will be of great assistance to practitioners in the control of these maladies. Further, they show that there is a close relation between certain infections of animals and public health. It has become very clear that one of the functions of the veterinary profession is to protect the human family against the dangers from meat and dairy food products and from the maladies of domesticated animals communicable to man. The saving of property and the safeguarding of human life provided by an efficient veterinary service fully justify the requests of the Dean for generous appropriations to continue the work and to increase the efficiency of the college in both teaching and research.

Respectfully,

LIVINGSTON FARRAND,
President.

LW
1924/25 cont.
dir 2
30 mar 26

TABLE OF CONTENTS

Letter of Submittal 3

Report of the Dean 7

Appendix:

Report of the Ambulatory Clinic 29
Report of the Surgical and Consulting Clinics 33
Report of the Small Animal Clinic 36
Report of the Diagnostic Work on Poultry Diseases 39
Report of the Diagnosis Laboratory 44
Autopsies: S. A. Goldberg and R. H. Volgenau 50
Vital Statistics of Diseases of the Genital Organs of Cows. D. H. Udall, E. R. Cushing and M. G. Fincher 52
Prevention of Diseases of Newborn Calves. D. H. Udall 67
Studies of Genital Disease in a Herd of Dairy Cattle. M. G. Fincher 79
The Channel of Invasion of Bacterium Abortum with Special Reference to Ingestion. R. R. Birch and H. L. Gilman 100
A Mould Associated with Abortion in Cattle . H. L. Gilman and R. R. Birch. 127
Some Common Diseases of Dogs. H. J. Milks 134
Bracken Poisoning of Cattle. W. A. Hagan 146
The Green Coloration Produced by Certain Streptococci on Blood Agar Plates. W. A. Hagan 152
Arteriosclerosis in Domestic Animals. S. A. Goldberg 163
A Clinical and Pathological Study of the Nervous Form of Canine Distemper. S. A. Goldberg and R. H. Volgenau 173
A Study of Skin Tuberculosis. C. M. Carpenter and S. A. Goldberg 190
Further Observations on the Blood and Urine of the Horse in Azoturia. C. E. Hayden 197
The Blood and Urine of the Cow in Milk Fever. A Second Report. C. E. Hayden 00
Case Report Illustrating the Value of the X-ray in Diagnosis. E. M. Curley 206
Fowl Plague. E. L. Brunett and S. Kondo 209
Immunizing Young Pigs against Hog Cholera. J. W. Benner 225
Experiments with Mixtures of Anti-hog Cholera Serum and Hog Cholera Virus. J. W. Benner 231

REPORT

ITHACA, N. Y., *July* 31, 1925.

President LIVINGSTON FARRAND:

SIR.—I have the honor to submit a report of the work of the New York State Veterinary College at Cornell University for the fiscal year ending June 30, 1925.

The veterinary college has operated in the usual way and with quite satisfactory results. The rapid development of new knowledge concerning the nature, prevention and treatment of animal diseases, renders the formulation of adequate courses of instruction an exceedingly difficult task. The extension of veterinary service into fields heretofore not benefited by it, the recent problems associated with poultry diseases and the increasing interest in the maladies affecting small animals, are making heavy demands on the teaching capacity of the college. They are indications that the course should be lengthened or the entrance requirements advanced to one or more years of college or university work. However, the veterinary situation in the country seems to indicate that for the present attention should be given to adjusting the new knowledge to the course as now given rather than expanding the curriculum. It is a matter of but a few years before additional preparation for the technical course will be necessary. It is recommended most earnestly that every student who can should obtain a thorough fundamental education before taking up veterinary medicine.

The veterinary service in the State is improving, although the small number of graduates is making it impossible for many communities, with large numbers of live stock, to secure resident practitioners, much as they are needed. There is a constantly increasing volume of veterinary work that should be done, but the long distance from practitioners compels the owners to withhold professional treatment from their sick animals that should have attention. Again, the poultry industry is expanding rapidly and there is much work to be done in the control of poultry diseases. The detecting of fowls infected with bacillary white diarrhea and removing them from the breeding flocks have become serious problems with many poultry owners. They are greatly in need of the technical advice and assistance that practitioners can and should be qualified to give. Tuberculosis is becoming a serious disease among fowls in many localities in this State and its detection and control present another task for the practitioner. In the poultry districts there is a real demand for professional service along these lines. It is important both for poultry owners and the veterinarian that it

should be rendered by duly qualified men. Further, small animal owners, especially in the cities, are availing themselves of veterinary service to a much greater extent than heretofore, and to meet these demands there must be more specialists in the diseases of small animals.

The regular veterinary work is not decreasing. The bovine tuberculosis eradication has now reached the point where there are many accredited herds to turn back to the local practitioners. The permanent success of the accredited herd plan, that is costing vast sums of public money, will depend upon the skill and leadership of local practitioners in keeping cattle owners interested in, and determined to have, tuberculosis free herds. Infectious abortion in cattle is spreading more or less rapidly and its control depends on the treatment and advice that is given by local veterinarians. The research work that has been done on this subject, and which has been published previously and in this report, points distinctly to the fact that the veterinarian is the man who must care for the aborting animal to see that she returns to regular breeding and to take the steps that are necessary to prevent the further spread of the infection and to maintain abortion free herds. The trouble can be dealt with successfully only on the farm by the veterinarian in cooperation with the owner.

The more recent discoveries of the communicability of certain infections of animals to man, such as *Brucella abortus* for cattle and the germs of tularemia and melioidosis of rodents, are pointing to the sanitary significance of an increasing number of animal infections. The campaign against bovine tuberculosis is causing tens of thousands of carcasses of reacting cattle to be placed on the market, which need very careful inspection. In many areas the importance to the community of obtaining competent veterinary service is being emphasized.

FACULTY

The college has been fortunate in being able to retain its teaching force, notwithstanding that attractive offers have come to several members of the staff. Dr. John M. Hendrickson, of Ohio State University, was engaged to assist in the study of poultry diseases. Dr. S. Kondo of Tokyo was given the courtesy of the laboratory through the year for the study of fowl plague and the problems involved in producing immunity against it.

As heretofore, the university furnished instruction to veterinary students in chemistry, embryology and histology and the College of Agriculture provided it in animal husbandry, botany and zoology. Reciprocally, the Veterinary College has given 461 university hours of instruction in physiology, bacteriology, pathology and horse shoeing to students in other colleges of the university. It also gave a special three-hour course the first term on "Health and Disease of Domesticated Animals" for students in the College of Agriculture, and Dr. Fish repeated his lecture course in physiology the second term for the same purpose.

PERSONNEL OF THE TEACHING STAFF

Livingston Farrand, president of the university.
Simon Henry Gage, emeritus professor of histology.
Walter Long Williams, emeritus professor of obstetrics and research in the diseases of breeding animals.

Department of Anatomy

Dr. Grant S. Hopkins, professor of anatomy.
Dr. Earl Sunderville, assistant professor.

Department of Materia Medica

Dr. Howard J. Milks, professor of therapeutics and small animal clinic.
Dr. H. C. Stephenson, instructor.

Department of Medicine.

Dr. Dennie H. Udall, professor of veterinary medicine and hygiene and superintendent of the ambulatory clinic.
Dr. M. G. Fincher, instructor.
Dr. E. R. Cushing, instructor.

Department of Pathology and Bacteriology

Dr. V. A. Moore, professor of comparative pathology and bacteriology and dean of the college.
Dr. W. A. Hagan, professor of bacteriology and parasitology.
Dr. S. A. Goldberg, assistant professor of pathology.
Dr. C. M. Carpenter, assistant professor of diagnosis.
Dr. E. L. Brunett, instructor in poultry diseases.
R. V. Gibbons, instructor in pathology.
Dr. J. M. Hendrickson, assistant in poultry diseases.
Dr. Frank Hare, assistant in diagnosis.
Mr. Charles J. Parshall, student assistant in diagnosis.
Mr. Alexander Zeissig, student assistant in bacteriology.

Department of Physiology

Dr. Pierre A. Fish, professor of veterinary physiology and secretary of the faculty.
Dr. C. E. Hayden, assistant professor.

Department of Surgery

Dr. J. N. Frost, professor of veterinary surgery.
Dr. M. H. Mabey, instructor.

Veterinary Experiment Station

Dr. Raymond R. Birch, superintendent of the station.
Dr. J. W. Benner, assistant professor.
Dr. H. L. Gilman, assistant professor.

Farriery

Mr. Henry Asmus, assistant professor of horseshoeing.

Library

Miss E. Clifford Williams, librarian of the Roswell P. Flower library.

Business Office

Miss Helena H. Haight, clerk and bookkeeper.
Miss Lillian M. Holden, stenographer and office secretary.
Miss Eileen Carey, clerk and stenographer.

Machinist

Mr. Archibald Wilson.

Thirteen men were employed as grooms, attendants and janitors. Some additional labor was required at irregular intervals, for which an appropriation was made.

NON-RESIDENT LECTURERS

The college was fortunate in securing lectures from Dr. Alexander Kotlan of the Royal Veterinary College of Hungary, this year exchange professor in America, on the life history of certain animal parasites; Dr. Theobald Smith, of the Rockefeller Institute for Medical Research, Department of Animal Pathology, Princeton, N. J., on research as applied to animal diseases; Dr. U. G. Houck of the Federal Bureau of Animal Industry, Washington, D. C., on the recent outbreaks of foot and mouth disease in the United States; and Dr. W. G. Hollingworth of Utica, N. Y., on the importance of municipal meat and dairy inspection. Drs. Charles E. Chase of Bay Shore, E. V. Moore of Cortland, E. L. Volgenau of Buffalo, and R. W. Gannett of Brooklyn, gave helpful talks on subjects pertaining to veterinary practice.

STUDENTS

For a number of years there has been a small registration in the veterinary colleges of the United States. The appended table, prepared by Dean White of Ohio State University, gives the number that matriculated in each state in 1923 and 1924. A few students entered at midyear, but presumably not more than the number of those who dropped out for various reasons.

Number of Students Attending Veterinary Colleges in the United States in 1923–24 *and* 1924–25

NAME OF COLLEGE	1923–1924						1924–1925					
	1st year	2d year	3d year	4th year	Sp.	Totals	1st year	2d year	3d year	4th year	Sp.	Totals
Alabama	3	2	4	7	.....	16	6	3	6	6	1	22
Colorado	10	15	12	13	1	51	6	5	14	11	.....	36
Georgia	2	8	7	5	.....	22	7	2	7	7	.....	23
Iowa	32	24	18	16	.....	90	27	27	17	17	1	89
Kansas	11	10	16	16	.....	53	20	8	11	16	5	60
Michigan	5	10	11	8	.....	34	11	7	10	10	2	40
New York	29	25	10	19	3	86	19	26	21	10	5	81
Ohio	13	12	22	20	5	72	7	7	23	30	2	69
Pennsylvania	14	10	10	5	2	41	25	10	10	10	1	56
Texas	11	4	5	2	.....	22	3	6	5	6	.....	20
Washington	8	8	4	6	.....	26	10	6	8	3	.....	27
Totals	138	128	119	117	11	513	141	107	132	126	17	523

In the appended table are given the number and value of the animals in the country on which are based the volume of veterinary service required and the number of veterinary students for a

period of ten years. This, however, does not include the 370,000,000 poultry, with an estimated value in excess of $500,000,000, or pet animals, for which we have no official estimate, either as to numbers or value. Each of these groups requires the services of many veterinarians.

Number and Value of Animals in the United States and the Number of Veterinary Students

YEAR	Number of animals	Value	Number of veterinary students
1914	190,583,000	$5,890,471,000	2,481
1915	198,577,000	5,969,253,000	2,608
1916	204,063,000	6,020,670,000	3,068
1917	205,637,000	6,835,602,000	1,250
1918	213,431,000	7,284,198,000	.....
1919	218,446,000	8,827,894,000	721
1920	190,679,000	8,164,573,000	903
1921	183,799,000	6,051,202,000	618
1922	184,316,000	4,799,170,000	716
1923	191,344,000	4,111,256,000	513
1924	194,441,000	4,842,673,000	523

INSTRUCTION

The facilities for teaching have been improved in many respects. The clinics have been well patronized, the number of cases exceeding those in previous years. This, in itself, increases the material for teaching practical medicine and surgery. To facilitate diagnoses an X-ray machine has been provided. It has proven to be of much value, especially in the diagnosis of fractures and tumors in both large and small animals. The extent to which it will be of practical value has not been determined, but it has been a most useful addition to the diagnosis equipment for the troubles mentioned.

The Ambulatory Clinic enables the senior students to come in contact with the conditions under which private practitioners encounter their patients. This clinic has charge of most of the medical work in a radius of eight miles or more from the college. Automobile transportation is provided for the students. The Surgical and Consulting Clinic receives many instructive surgical cases from outside our regular territory. These are usually important cases and exceptionally valuable for teaching purposes. The necessity for more room in the surgical hospital will be met by the addition of a second story to the present building. The contract for this has been awarded and it will be ready for occupancy late in the fall. The Small Animal Clinic continues to show a satisfactory growth. The diseases of pet animals that are

brought there include practically all those which occur in these species in this latitude. Like the Surgical Clinic, many cases are brought to it from some distance away. The facilities for teaching the diseases of small animals are excellent and material for clinic instruction is abundant. The actual number of cases treated in each of the clinics during the year is given in the appended table.

Clinic	*Number of Cases*
Ambulatory and medical	3,782
Surgical and consulting	1,371
Small animal	1,638
Total	6,791

In addition to the clinics, there are four other distinct activities in the college of direct value to students in the upper classes. They are the post mortem examinations, in which the senior students participate; the work in the diagnosis laboratory; the work in the diagnosis and study of poultry diseases; and the preparation of anti-hog cholera serum. The volume of work in each of these services for the year is indicated in the appended table.

Post mortems	284
Diagnosis laboratory (number of specimens examined)	2,416
Tuberculin made and distributed to veterinarians (doses)	52,446
Autogenous vaccines and bacterins (mils)	21,850
Poultry specimens for diagnosis	646
Anti-hog cholera serum distributed (mils)	278,120
Hog cholera virus for immunizing purposes (mils)	8,612

These activities offer exceptional opportunities for graduate and senior students who become interested in these phases of professional work.

The teaching of horseshoeing to veterinary students is becoming more important than heretofore. The farriery is well equipped. The treatment of the troubles in the feet of horses that can be remedied only by proper shoeing must be directed by the veterinarians themselves more than formerly, as trained horseshoers are rapidly disappearing. It is not unlikely that veterinarians will be obliged often to adjust the shoes, for many shops to which such cases could be sent in the past are closed.

The short courses in horseshoeing that were started with much promise before the war are no longer patronized because of the lack of men taking up this work. The farmers are obliged in many sections to do their own shoeing and, consequently, short courses in horseshoeing are being given to farmers in connection with the extension instruction in farm mechanics from the College of Agriculture. In this work Professor Asmus has been very successful and the demand for instruction on the part of horse owners is increasing rapidly. It is fortunate that the college has a

skilled shoer who can do this work. In addition to this a special course in horseshoeing is given for students in the College of Agriculture.

The artillery unit of the Reserve Officers' Training Corps, located at Cornell University, furnishes considerable experience in the treatment of equine diseases. The veterinary unit is still maintained under the immediate supervision of Captain E. M. Curley of the Veterinary Corps.

The South Wing to James Law Hall has been completed and equipped. It has provided adequate office room, an amphitheatre, a satisfactory diagnosis laboratory and a library with spacious stack and reading rooms. These have increased greatly the facilities of the college and given it a completeness that has not existed heretofore. The amphitheatre provides a suitable place for the annual veterinary conference and other large gatherings.

LIBRARY

The library is now well housed in the South Wing of James Law Hall. A large reading room with ample reference shelves already has increased its usefulness by at least 50 per cent. There are now in the library 7,272 volumes, and 95 veterinary and medical journals and those of closely allied sciences are received. There were 328 volumes added this year. We wish to acknowledge with much appreciation and thanks the gift of several interesting volumes from Professor George H. Burr of Cornell University, Drs. H. D. Martin and E. L. Volgenau of Buffalo, and Dr. B. D. Pierce of Springfield, Mass. The library is indebted also to a number of other friends who have donated one or more books of special interest to the veterinary profession. The librarian is arranging, as rapidly as possible, to extend the use of the library to the practitioners in the State. The rapid development of veterinary and biological sciences has increased the demand for a larger number of scientific journals. It is to be regretted that the library funds are inadequate for this purpose.

VETERINARY CONFERENCE

The seventeenth annual conference for veterinarians of the State was held January 15th and 16th. The attendance was the largest in the history of these gatherings. Deep interest was exhibited in the subjects presented. The program contained papers of immediate practical value to practitioners, which were given by men who stand foremost in their experience with the subjects. The increasing numbers that attend these conferences give undisputed evidence of their value to the practitioners. The new amphitheatre with its moving picture machine and balopticon made it possible to present certain subjects in a much more effective manner than heretofore. The conferences have come to be occasions, not only for giving and receiving technical information, but opportunities for clearing up differences that may have arisen relative to dairy

inspection, official tuberculin testing and other professional work. The animal owners of the State are fortunate in having a body of veterinarians so deeply interested in the progress of knowledge on animal diseases and their prevention. Most of the papers presented were printed in the April number of the Cornell Veterinarian and a copy was sent to each practitioner of the State. A list of the papers presented at the conference is appended:

Address of welcome.....President Livingston Farrand
Outlook for veterinarians, determined by the number of students, number of animals and their value.....V. A. Moore
A mould associated with abortion in cattle....H. L. Gilman and R. R. Birch
Channels of infection by Bacterium abortum, with special reference to ingestion.....R. R. Birch and H. L. Gilman
Vital statistics from the ambulatory clinic.....
D. H. Udall, M. G. Fincher and E. R. Cushing
Tuberculin testing by accredited veterinarians....Dr. Charles Linch, Department of Farms and Markets, Albany, N. Y.
A study of skin tuberculosis (Lantern slides)..C. M. Carpenter and S. A. Goldberg
The endocrine system and growth.....P. A. Fish
The significance of colostrum in the prevention of the diseases of young calves.....Theobald Smith, Rockefeller Institute for Medical Research, Princeton, N. J.
The viscera of the dog.....E. Sunderville
Lesions in the nervous form of canine distemper (Lantern slides) S. A. Goldberg
Some common diseases of dogs (Lantern slides).....H. J. Milks
Rabies and its control.....V. A. Moore
Foot and mouth disease (Film).....U. G. Houck, Federal Bureau of Animal Industry, Washington, D. C.
The veterinarian and organized agriculture.....Charles H. Tuck, Dairymen's League, New York City
The veterinarian's responsibility to his community..Garry T. Stone, Binghamton, N. Y.
The economic value of the poultry industry.....Prof. James E. Rice
Poultry Diseases—
(a) Diagnosis of bacillary white diarrhoea
(b) A new disease of chickens in America (fowl plague)..E. L. Brunett
Surgical treatment of indigestion in cattle.....J. N. Frost
Surgical diagnosis..John W. Adams, University of Pennsylvania, Philadelphia, Pa.
The future of veterinary surgery.....John W. Adams
Opportunities for veterinarians in the South....J. G. Ferneyhough, State Veterinarian, Richmond, Va.

DEMONSTRATIONS

Amplification of heart beat sounds.....P. A. Fish
Pathological specimens of some of the diseases of small animals.. S. A. Goldberg
Method of collecting blood from fowls for agglutination test in bacillary white diarrhoea.....E. L. Brunett
Preparation of tuberculin.....C. M. Carpenter
Small animal clinic.....H. J. Milks and H. C. Stephenson
Demonstration in the methods used in the study of the diseases of the genital organs..W. L. Williams, D. H. Udall, E. R. Cushing and M. G. Fincher
Shoeing for lameness.....H. Asmus
Clinic and dressing of cases.....J. N. Frost and M. H. Mabey
Method of drawing blood for agglutination test as applied to abortion disease.....R. R. Birch and H. L. Gilman

At the evening session Dr. John McCartney, president of the association of alumni of this college, presented to the university, on behalf of the alumni, an excellent portrait of Dr. P. A. Fish, painted by Professor O. M. Brauner. It was very cordially accepted by President Farrand.

LABORATORY DIAGNOSIS.

The amount of diagnostic work requested by the practitioners and animal owners has increased steadily for several years. Associated with the diagnosis there has been a growing interest in the use of autogenous vaccines and bacterins, more especially for infectious mastitis in cattle. The results indicate that there is some virtue in such products. This co-operative work with the practitioners promises to be of much assistance not only to them but to cattle owners themselves. There has been a smaller demand for tuberculin, as would be expected, since the official state tests are made with tuberculin furnished by the Federal Bureau of Animal Industry.

There is a growing demand for laboratory work that has passed beyond the experimental stage and come to be a matter of routine. The agglutination tests for infectious abortion and for bacillary white diarrhoea in chickens are good illustrations of this point. They are recognized procedures required in the diagnosis and disease control work of practitioners. The appropriations by the State for research are not available for routine service, which has become part of practice and should be done either by the practitioners or in private laboratories. If there is no other way by which such important examinations can be made, they should be provided for in the college diagnosis laboratory on a fee basis, thereby providing funds for the employment of assistants. The efficiency of veterinary service is assured to a large degree by the extent practitioners accept and use the newer methods of diagnosis, treatment and control. Much of this work can be done by practitioners with a small outlay for equipment. If all material that requires microscopic examination is sent to a distant laboratory for determination, the owners are not impressed with the efficiency of their advisers. Veterinarians should be capable of making at least the more common determinations and, wherever it is possible, the practitioner will find it to his advantage to do the work. It is the application of methods of precision that gives them standing as professional men in the community. Hence the desirability of individuals applying as many of the scientific methods and making as many examinations as possible themselves, leaving the laboratory available for such work as cannot be done in the field. Laboratory courses in bacteriology, pathology, and blood and urine analyses are provided in the curriculum to teach men how to make the simpler and more common examinations required in routine practice.

RESEARCH

The research work that was under way has been continued and new studies have been undertaken. The funds available for this purpose are not sufficient to carry on investigational work and research in animal diseases as rapidly as their importance warrants. Several problems are being studied as material permits. The researches on the breeding diseases of cattle are receiving most attention because of their great economic and, possibly, sanitary significance, and further because our research funds are appropriated largely for this purpose. The mystery of an animal disease is often difficult to penetrate quickly, and years may be necessary to determine the essential facts relative to its nature. The work on the subjects appended has been carried to the point where the authors feel that it should be recorded.

In calling attention to research on animal diseases, I am constrained to emphasize the importance of utilizing in a practical manner the knowledge already available. It is in this way only that animal husbandry will profit by the expenditures for research. Often we are chagrined by the heavy losses due to the fact that practitioners and official veterinarians have neglected to utilize existing knowledge to diagnose and check outbreaks and epizootics that should be controlled readily. The application of demonstrated and verified truths relative to the nature of a disease is a practical corollary to research. To accomplish that feat is the ambition of many teachers who devote their time and energies to the most difficult of all problems, effective teaching. The results of such efforts will be recorded in more efficient practice and live stock sanitation.

The appendix will contain reports on the following subjects:

The clinics, post mortems, diagnosis laboratory and poultry disease investigation.
Vital statistics of diseases of the genital organs of cows.
The prevention of diseases of new-born calves.
Studies of genital diseases in a herd of dairy cattle.
The channel of invasion of *Bacterium abortum* with special reference to ingestion.
A mould associated with abortion in cattle.
Some common diseases of dogs.
Bracken poisoning of cattle.
The green coloration produced by certain streptococci on blood agar plates.
Arteriosclerosis in domestic animals.
A clinical and pathological study of the nervous form of canine distemper.
A study of skin tuberculosis.
Further observations on the blood and urine of the horse in azoturia.
The blood and urine of the cow in milk fever.
Case report illustrating the value of the X-ray in diagnosis.
Fowl plague.
Immunizing young pigs against hog cholera.
Experiments with mixtures of anti-hog cholera serum and hog-cholera virus.

Reports of Clinics. These reports give the number of animals examined, the diseases from which they suffered and the results of the treatment. They indicate clearly the facilities of the college for teaching veterinary medicine and surgery.

Autopsies. This is a report of 284 post mortems by Dr. Goldberg. It gives the disease from which the animals suffered and it shows also the abundance of material for experience in making autopsies and the study of gross and microscopical pathology. These records, which now exceed 2,000 in number, contain valuable information on the secondary morbid changes as well as the primary causes of death.

Diagnosis Laboratory. This laboratory, which began by making a few diagnoses for practitioners, has increased in its activities until it now requires the time of two persons to meet the demands. There is considerable research suggested by the problems encountered, but, unfortunately, there is time for carrying out but very few of them. This year the number of examinations was 2,416. Skin tuberculosis and the study of *Brucella abortus* in market milk and in a case of undulant fever in man have received considerable attention. This report goes into details relative to the examinations for rabies, anthrax and other specific infectious diseases encountered in this State by veterinarians. It includes also the results of autogenous vaccines and bacterins in the treatment of mastitis and a statement of the quantity of biologics made and distributed at cost to practitioners. It is of interest to note that there is considerable demand from silver fox owners for aid in connection with the diseases of foxes.

Poultry Diseases. The report of Drs. Brunett and Hendrickson on the diagnosis of poultry diseases should be of much interest to veterinanians residing in poultry districts. The losses from bacillary white diarrhea are becoming so heavy that the control of this infection is of much economic importance. Veterinarians can be very useful in the control of this malady and also in diagnosing the other infections, such as chicken cholera, tuberculosis and fowl plague. The heavy investment in poultry calls for careful consideration of their diseases and the necessity of the veterinarians including the feathered tribes among their prospective patients.

Vital Statistics of Diseases of the Genital Organs of Cows. In this report Drs. Udall, Cushing and Fincher record the findings in a thousand cases of genital organ troubles in cattle met with in their clinical work from July, 1920, to December, 1924. This careful analysis of so many cases, occurring in numerous herds and under different conditions, reveals a fund of information on these troubles impossible to obtain otherwise. The data should be of great interest and help to practitioners.

Prevention of Disease of New-born Calves. The purpose of this study was to determine the best method of handling new-born calves in herds where losses from disease occurred. Calves often sicken and die when but a few days old, with or without symptoms of scours. The hypothesis on which the study was undertaken was "improper feeding and not infection" as the cause of the trouble. Working on this theory Dr. Udall has formulated a method of feeding calves that he has found to be very satisfactory. Every veterinarian who has worked among dairy cattle will appreciate the instructions based on the experience contained in this article.

Studies of Genital Diseases in a Herd of Dairy Cattle. This is a detailed report of two herds treated in the Ambulatory Clinic that have been under observation for some years. The data presented and the summary made by Dr. Fincher will be of much value to practitioners. They contain information that emphasizes the danger to healthy pregnant cattle of spreaders of the Bang organism.

The Channel of Invasion of Bacterium Abortum with Special Reference to Ingestion. Among the micro-organisms associated with the breeding troubles in cattle, the one described by Bang is of first importance. It has long been a question whether infection with this micro-organism occurs through both the mouth and the genital tract, or through the genital tract alone, and this doubt, or acceptance, of the genital tract as the one channel of infection has led many veterinarians and cattle men to ignore precautions to prevent association of pregnant cows with spreaders of *Brucella abortus,* the cause of the disease. Experiments carefully planned and carried out by Drs. Birch and Gilman are described in this paper. They indicate clearly that the infection of pregnant cows, terminating in abortion, may be brought about readily by feeding them cultures of the Bang organism. The experiments show clearly the importance of preventing sound, pregnant cows from associating with spreaders of this organism.

A Mould Associated with Abortion in Cattle. This paper by Drs. Gilman and Birch describes the finding of a *mould* as the probable cause of abortion in a few cattle. Since the discovery by Bang of the organism of infectious abortion, a number of other micro-organisms have been incriminated as the cause of breeding troubles. Some of these are of relatively infrequent occurrence, but it is important to recognize them when they appear, and to ascertain the part they play in producing genital disease. The mould here described was isolated from three cases of abortion, cultivated artificially and shown to be capable of producing abortion in sound, pregnant cattle.

Some Common Diseases of Dogs. This paper by Dr. Milks has for its purpose primarily to emphasize the more important of the common ailments of this species that practitioners encounter. It will be of interest to those having a canine practice.

Bracken Poisoning of Cattle. For a number of years a disease of undetermined etiology has appeared in cattle in the late summer and fall. In some localities and in certain years the losses from it have been quite serious. Dr. Hagan has studied a number of cases. More recently his attention has been directed to the possibility of bracken poisoning as the cause. The information given will assist those who may encounter this, as yet, somewhat mysterious disease.

The Green Coloration Produced by Certain Streptococci on Blood Agar Plates. This is a technical paper on the study of green coloration about the colonies of certain streptococci, and, possibly, other green producing bacteria. Dr. Hagan has found

that it is due to the action on the blood corpuscles of peroxide and acid produced by these organisms when in active multiplication. This will be of interest to those who are studying streptococcic infection.

Arteriosclerosis in Domestic Animals. Arteriosclerosis is quite prevalent in the human subject above middle age, where it is often claimed to be a senile affection. In animals this condition is not so well known. The report by Dr. Goldberg contains valuable data on this subject. He has made a careful study to determine whether the arteriosclerosis that occurs in animals is similar to that found in man. He concludes that in some respects it is similar, but not identical.

A Clinical and Pathological Study of the Nervous Form of Canine Distemper. Canine distemper is perhaps the most destructive disease of dogs, the nervous form being the most fatal. Although numerous investigators have worked on this disease, there are still many undetermined factors in connection with it. In this report Drs. Goldberg and Volgenau have made a careful study of the pathology of the disease, or more especially the nervous form. The work was begun in 1916, and many of the lesions have been determined. The report will be of special interest to those interested in canine diseases.

A Study of Skin Tuberculosis. In the eradication of bovine tuberculosis it has been found frequently that cattle react to tuberculin after several negative tests. In these animals it sometimes happens that the only lesion found is in the skin. Drs. Carpenter and Goldberg undertook this investigation to determine whether these lesions were true tuberculosis or changes due to other acid-fast bacteria. The findings indicate that the lesions studied were due to tubercle bacteria and not to other organisms of the acid-fast group.

Further Observations on the Blood and Urine of the Horse in Azoturia. This is a report of a careful analysis of the blood and urine in a number of cases of azoturia for the purpose of determining (1) the actual composition of these fluids during the attacks, and (2) to ascertain if there is justification for the claim that this disease is due to disturbed carbohydrate metabolism. Dr. Hayden has done much work on this subject and his earlier findings have been recorded in previous reports. In the case here detailed, newer methods have been employed. He finds that non-protein nitrogen and its component parts, urea, uric acid and creatinine, are very close to the normal, and that there is no marked nitrogen retention. He also finds that there is no justification for the assumption that azoturia is due to disturbed carbohydrate metabolism.

The Blood and Urine of the Cow in Milk Fever. This report by Dr. Hayden is a continuation of the study of this subject in which he has presented considerable new and valuable data on the chemistry of the blood and urine of cattle affected with this trouble.

Case Report. Captain Curley has presented a most interesting case in which the use of the X-ray in diagnosis is brought out very clearly. It will be of interest to all practising veterinarians.

Fowl Plague. This report by Drs. Brunett and Kondo contains a description of cases of fowl plague that appeared in this State in the fall of 1924. As it is the first time this destructive disease has been identified in the United States, its description and the localities where it appeared will be of unusual interest. It has been estimated that this disease, and those mistaken for it, caused the loss of more than a half million fowls in the winter of 1924–1925 in the New York markets alone.

Immunizing Young Pigs against Hog Cholera. This report by Dr. Benner contains the results of experiments that have been carried out this year in immunizing young pigs. It is a continuation of the work started some years ago with the hope that the cost of immunizing against hog cholera could be reduced. The results are encouraging.

Experiments with Mixtures of Anti-hog Cholera Serum and Hog Cholera Virus. Dr. Benner has carried out a series of experiments to ascertain if it is possible to mix the serum and virus before its injection, thereby minimizing the labor involved in the simultaneous method. The preliminary work here reported is not especially encouraging, and for the present the procedure cannot be recommended.

NEEDS OF THE COLLEGE

The outstanding need of the college is the laboratory building for pathology, bacteriology and research in animal diseases already requested of the Legislature. The present post mortem room is inadequate, and the need of an incinerator is obvious. A building for experimental animals is very much needed. The heavy losses among live stock from disease warrant much greater expenditures for research and the preparation of veterinarians. New diseases are developing and the protection of the animal husbandry of the State against the losses they occasion rests in a properly distributed and efficient veterinary profession. The State is now paying millions of dollars annually to eradicate bovine tuberculosis, while in states that maintain competent veterinary supervision the cost is very small. Such diseases as infectious abortion and Johne's disease in cattle and several destructive maladies of poultry require much research before their nature is understood fully, and before the losses caused by them can be minimized.

APPROPRIATIONS

The Legislature of 1925 appropriated $137,020 for the maintenance of the college for the year 1925–26. This is $8,160 more than the amount allowed in the preceding year. There were no appropriations for new construction.

RECOMMENDATIONS

It is recommended that the trustees request of the Legislature of 1926 a sum sufficient for maintenance, to increase the research work on animal diseases, make the necessary repairs and just and deserved adjustments in salaries. There is a great need for a post mortem room and incinerator. These are planned as a part of the new laboratory building for bacteriology, pathology and research, which it is hoped will soon be provided.

PUBLICATIONS AND ADDRESSES OTHER THAN THOSE INCLUDED IN THE ANNUAL REPORT BY MEMBERS OF THE FACULTY

BIRCH, R. R.

Hog Cholera and Its Complications. Address, Conference for veterinarians at New Brunswick, N. J., February 26, 1925.

Breeding Problems. Address, Local Guernsey Cattle Club, Elbridge, N. Y., June 19, 1925.

Hog-Cholera Problems as They Relate to the Practitioner. Address given at the meeting of the Kentucky Veterinary Medical Association in July, 1924. *Journal of the American Veterinary Medical Association,* January, 1925.

BRUNETT, E. L.

Poultry Diseases. *Cornell Veterinarian,* Vol. XIV, p. 89.

The Occurrence of a Disease of Chickens in New York State caused by a Filterable Virus. *Journal Am. Vet. Med.* New Series, Vol. XIX, p. 497.

A Filterable Virus Disease of Chickens (Fowl Plague). *Cornell Veterinarian,* Vol. XV, 1925, p. 4.

The Diseases Prevalent among Poultry in New York State. Address, Farmers Week, Canton, N. Y., December, 1924.

Poultry Sanitation and Disease Diagnosis. Address, Farmers Week, Ithaca, N. Y., 1925.

Internal Parasites of Poultry. Address, Farmers Week, Ithaca, N. Y., 1925.

Fowl Plague. Address, Poultry meeting, Auburn, N. Y., January, 1925.

Bacillary White Diarrhea. Paper, Veterinary Conference, Ithaca, 1925.

Clinical and Post Mortem Diagnosis and Prevention of So-called European Fowl Plague. Significance, distribution, etc. Address, Amherst, Mass., May, 1925.

Description of Cases of Chicken Cholera, Chicken Pox and Constitutional Diseases. Address, Amherst, Mass., May, 1925.

Fowl Plague and Attempts at Immunization. Paper, Annual meeting of N. Y. State Vet. Med. Soc., at Ithaca, N. Y., June, 1925.

Respiratory Diseases of Poultry and Fowl Plague. Address, Poultry meeting, White Plains, N. Y., 1925.

FISH, P. A.

The Wright Curves of Castrated Kids. *Proceed. Soc. Exp. Biol. & Medicine,* Vol. XXII, 1925, pp. 248–250.

Castration, Nutrition and Growth. *North American Veterinarian,* Vol. VI, 1925, pp. 21–43.

The Endocrine System and Growth. *Cornell Veterinarian,* Vol. XV, 1925, pp. 160–172.

GOLDBERG, S. A., MAYNARD, L. A. and MILLER, R. C.

The Influence of Sunlight on the Mineral Nutrition of Swine. *Proc. Soc. Exp. Biol. & Med.,* May, 1925, xxii, p. 494.

GOLDBERG, S. A. and SIMPSON, S.

Certain Pathological Tissue Changes in Thyroidectomized Sheep. *Proc. Soc. Exp. Biol. & Med.,* 1924, xxi, 567.

Arteriosclerosis in Thyroidectomized Sheep. *J. Med. Res.,* 1924, xliv, 669.

GOLDBERG, S. A., WILLIAMS, W. L., UDALL, D. H., FROST, J. N., et al.
See Williams et al.

GOLDBERG, S. A., MAYNARD, L. A. and MILLER, R. C.
A Study of the Dietary Relationships and the Pathology of "Stiffness" in Swine. Cornell University Agr. Exp. Sta., Mem. 86, February, 1925.

GOLDBERG, S. A.
The Basis of symptoms in the Nervous Form of Canine Distemper. Paper, Conference of Veterinarians, Univ. of Penn. School of Vet. Med., January 6, 1925.
A discussion of Chronic Arthritis. Paper, Medical Staff of the Geneva General Hospital, Geneva, N. Y., December 11, 1924.
Arteriosclerosis in Thyroidectomized Sheep. Paper, Tompkins Co. Medical Society, Ithaca, N. Y., November 18, 1924.

HAGAN, W. A. and MOORE, V. A.
See Moore and Hagan.

HAYDEN, C. E.
Seventeenth Annual Conference for Veterinarians at Cornell University. *Jour. Am. Vet. Med. Assoc.*, 1925, lxvi, 779–782.

MILKS, H. J.
Drugs in Disease. Paper presented before the N. Y. County Vet. Med. Soc., November, 1925. *Cornell Veterinarian*, Vol. XV, No. 1, p. 52, January, 1925.
Small Animal Practice. Paper, joint meeting of Vermont and New Hampshire Vet. Med. Societies, Lakeport, N. H., September 5, 1924.

MOORE, V. A.
Fowl Plague, a new Chicken Disease in America. *The Cornell Veterinarian*, Vol. XV, 1925.
Rabies and its Control. *Proceedings U. S. Live Stock Sanitary Assoc.*, 1924.
Some Problems for Veterinarians. *Jour. Am. Vet. Med. Assoc.*, 1925, p. 75.
The Veterinary College at Cornell University. *Dairyman's League News*, Vol. IX, 1925, p. 18.
Veterinary Education. Bulletin No. 9, Dept. Interior—Bureau of Education, 1925.
The Outlook for Veterinarians. *The Cornell Veterinarian*, Vol. XV, p. 75.
Poultry Diseases and Their Control. Address, Mass. State Vet. Society, Amherst, Mass., May, 1925.
Meat Inspection and Its Relation to Public Health. Two lectures, Mass. Inst. Tech., April, 1925.
The Prevention of Fowl Plague. Sent to Poultry Journals, Farm Bureau News and Farm Papers.
Bovine Tuberculosis Control. (Proceedings of the World's Dairy Congress, 1923. Published, 1924.)

MOORE, V. A. and HAGAN, W. A.
Laboratory Manual in General and Pathogenic Bacteriology and Immunity. Ginn & Co., pp. XXII–252. 1925.

SUNDERVILLE, E.
The Viscera of the Dog. Paper, Veterinary Conference, Ithaca, 1925.

UDALL, D. H.
Four lectures. Post-Graduate Short Course for Veterinarians, Michigan Agricultural College, January, 1925.
1. The Care and Feeding of New-born Calves.
2. Lead Poisoning and Indigestion in Calves.
3. The Relation of Sound Genital Organs to Healthy Cows.
4. Diseases of the Udder and Feet.

Diseases of the Digestive System of Cattle. *Proceedings of the World's Dairy Congress*, 1924, Vol. 2, p. 1460.
Studies in Genital Disease. *Cornell Veterinarian*, Vol. XIV, 1924.

WILLIAMS, W. L., UDALL, D. H., FROST, J. N., GOLDBERG, S. A., CUSHING, E. R,. FINCHER, M. G. and MABEY, M. H.
Studies in Genital Disease. *Cornell Veterinarian*, XIV, 315.

FINANCIAL STATEMENT

REPORT OF THE CIRCULATING FUNDS OF THE NEW YORK STATE VETERINARY COLLEGE AT CORNELL UNIVERSITY FROM JULY 1, 1924, TO JUNE 30, 1925

Balance July 1, 1924		$8,070 33
Receipts 1924–1925:		
Tuition	$1,490 00	
Laboratory fees, regular	2,405 95	
Laboratory fees, special	45 00	
Clinics	9,210 26	
Horseshoeing	1,390 76	
Tuberculin and bacterins	2,072 34	
Serum and vaccine	3,762 47	
Sale hogs and pork	1,814 72	
Sale other animals	740 14	
Sale apparatus, supplies, etc	421 34	
Rebates and dividends	102 42	
Rent	129 00	
		23,584 40
Disbursements 1924–1925:		
Summer work	$2,625 00	
Labor and overtime	944 15	
Night and Sunday work	542 50	
Lecturers	300 00	
Clerical	370 70	
Drawing and sewing	388 60	
Outside calls	90 00	
Laundry	306 89	
Equipment	2,340 28	
Supplies	7,210 39	
Materials	105 65	
Communication	678 85	
Printing and advertising	1,598 95	
Traveling and hotel	959 72	
Automobile	2,798 98	
Animals	2,052 98	
Repairs	532 64	
Fuel, light, heat, water	497 53	
Insurance and taxes	654 26	
Permanent improvements	219 75	
Miscellaneous	39 52	
	$25,257 34	
Balance July 1, 1925	6,397 39	
	$31,654 73	$31,654 73

EXPENDITURE OF THE MAINTENANCE FUNDS FOR THE NEW YORK STATE VETERINARY COLLEGE FOR THE YEAR 1924–1925

(Chapter 140, Laws of 1924)

Administration, salaries	$11,340 00
Teaching and research, salaries	67,550 00
Care of plant, wages	13,020 00
Temporary labor	1,598 13
Heat, light, water, power	5,500 00
Equipment, supplies and materials	7,250 00
Traveling expenses	498 10
Communication	1,249 99
Printing	2,013 13
Repairs	1,717 80
Investigation of animal diseases	9,999 06
Investigation of poultry diseases	6,836 12
	$128,572 33
Balance to be returned to the State	287 67
	$128,860 00

EXPENDITURE OF THE SPECIAL FUNDS FOR THE NEW YORK STATE VETERINARY COLLEGE FOR THE COLLEGE YEAR 1924–1925

Balances July 1, 1924:			
Addition to laboratory building (Chapter 140, 1924)			$98,923 74
Equipment and furniture (Chapter 225, 1923)			17,585 01
Serum laboratory (Chapter 225, 1923)			1,131 27
Repair of parapet (Chapter 225, 1923)			1,788 72
Appropriation for surgical ward (Chapter 140, 1924)			37,000 00
Expenditures 1924–1925:			
Addition to laboratory building		$77,025 37	
Equipment and furniture		17,424 86	
Serum laboratory		1,117 45	
Repair of parapet		648 41	
Surgical ward		66 99	
		$96,283 08	
Balances July 1, 1925:			
Addition to laboratory building	$21,898 37		
Equipment and furniture	160 15		
Serum laboratory	13 82		
Repair of parapet	1,140 31		
Surgical ward	36,933 01		
		60,145 66	
		$156,428 74	$156,428 74

As per itemized vouchers approved by the State Comptroller.

H. H. HAIGHT,
Clerk.

The appendix, which contains the reports of the clinics, diagnosis laboratories and reports of research by members of the faculty, is an integral part of this report. The faculty has cooperated to the end that the college shall perform fully its duties and meet its obligations to its students, to the university and to the State.

Respectfully submitted,

V. A. MOORE,

Dean, New York State Veterinary College at Cornell University.

APPENDIX

Containing Reports of the Clinics and Research

REPORT OF THE AMBULATORY CLINIC

From July 1, 1924, *to June* 30, 1925

D. H. UDALL, E. R. CUSHING, AND M. G. FINCHER

Department of Veterinary Medicine, Obstetrics, and Ambulatory Clinic

The attached report of the animals treated in the Ambulatory Clinic during the year ending June 30, 1925, shows a total of 3,782 cases divided as follows:

Cattle	2480
Swine	681
Horses	398
Sheep	223
Total number of calls	3782

This reveals a marked increase in the number of cattle treated and examined. A part of the increase is explained by the fact that dairymen are gradually coming to realize the value of treatment of cows that do not breed readily, that abort, have retained placenta, or show other signs of metritis. Increased requests for physical examinations and the tuberculin test, to meet the increasing demands of the public, account for larger numbers each year. There was a slight decrease in the number of sheep and horses treated, and a decrease of 43 per cent in the number of swine.

As in previous years, material for laboratory examination has been collected, and the reports filed with the clinical records. It is believed that this extension of our clinical knowledge will contribute materially to a more complete understanding of the cases studied.

Whenever information of sufficient importance to justify a report is assembled, an effort is made to publish it. During the past few months the following paper was printed in addition to those included in this report.

Studies in Genital Disease[1] (Joint Authorship)

For the most part this is a description of lesions observed in the placentae, and their correlation with the corresponding patient. A considerable part of the material and records came from the ambulatory clinic. It was only through joint effort that the collection of the material, its study and description when fresh, and its complete record could be made possible. This report is of chief value to clinicians for the purpose of estimating the corresponding lesions in the uterus. In this respect I believe it to be original. It has been clearly shown that a careful study of the afterbirth may reveal the history, the pathological condition, and the probable breeding future of the patient. Further observations are necessary to establish the permanent value of this method of diagnosis and prognosis.

[1] Williams, W. L., Udall, D. H., Frost, J. N., Goldberg, S. A., Cushing, E. R., Fincher, M. G., and Mabey, M. H. Studies in Genital Disease, *The Cornell Veterinarian*, 1924, xiv, 315–374.

CASES TREATED IN THE AMBULATORY CLINIC

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Killed or died	Undetermined
1. INFECTIOUS DISEASES									
Actinomycosis	6	....	....	....	1	2	....	3	.....
Diseases of the new-born	....	7	2	....	7	....	....	2	.....
Hemorrhagic septicemia	....	....	12	....	12	....	....	....	.....
Hog cholera	....	....	....	2	2	....	....	....	.....
Malignant head catarrh	....	1	....	....	....	....	....	1	.....
Necrobacillosis	....	1	....	7	7	....	....	1	.....
Purpura hemorrhagica	1	....	....	....	....	....	....	....	1
Tetanus	1	....	....	....	1	....	....	....	.....
Tuberculosis	....	6	....	....	....	....	....	6	.....
2. DISEASES OF THE CIRCULATORY SYSTEM									
Azoturia	8	....	....	....	7	....	....	1	.....
Dilatation of heart	1	....	....	....	....	....	....	1	.....
Lymphangitis	19	....	....	....	17	1	....	1	.....
3. DISEASES OF THE RESPIRATORY SYSTEM									
Congestion of lungs	1	....	....	....	1	....	....	....	.....
Heaves	5	....	....	....	....	4	....	....	1
Hemoptysis	1	1	....	....	....	....	....	2	.....
Laryngitis	5	1	....	....	6	....	....	....	.....
Pharyngitis	3	3	....	....	6	....	....	....	.....
Pneumonia	2	6	....	....	2	....	....	6	.....
4. DISEASES OF THE DIGESTIVE SYSTEM									
Choke	2	7	....	....	8	....	....	1	.....
Enteritis	....	5	....	....	3	....	....	2	.....
Esophagitis	....	1	....	....	1	....	....	....	.....
Foreign body in mouth	....	1	....	....	1	....	....	....	.....
Gastro-enteritis	....	2	....	....	1	....	....	1	.....
Gastro-intestinal catarrh	17	9	....	....	26	....	....	....	.....
Hepatitis	....	1	....	....	....	....	....	1	.....
Impaction of abomasum	....	1	....	....	....	....	....	1	.....
Impaction of colon	11	....	....	....	8	....	....	2	1
Incarceration	1	....	....	....	....	....	....	1	.....
Indigestion	66	77	....	4	138	....	....	6	3
Intussusception	1	1	....	....	....	....	....	2	.....
Parasitism, ascariasis	9	....	....	....	8	....	....	....	1
Parasitism, stomach-worm disease	....	....	100	....	50	....	....	....	50
Parasitism, strongylidosis	10	....	....	....	3	2	4	....	1
Prolapse of rectum	....	....	1	....	....	....	....	1	.....
Rupture, cecum	1	....	....	....	....	....	....	1	.....
Rupture, stomach	1	....	....	....	....	....	....	1	.....
Teeth, long	2	....	....	....	....	....	....	....	.....
Teeth, sharp	53	....	....	....	....	....	....	....	.....
Teeth, split	9	....	....	....	....	....	....	....	.....
Torsion, colon	2	....	....	....	....	....	....	2	.....
Torsion, small intestine	1	....	....	....	....	....	....	1	.....
Traumatic gastritis	....	25	....	....	....	2	4	13	6
5. DISEASES OF THE GENITO-URINARY SYSTEM									
Aberration, genitals	....	2	....	....	....	....	....	2	.....
Abortion	....	99	....	....	95	....	....	3	1
Balanitis	....	1	....	....	....	1	....	....	.....
Castration	5	1	28	38	....	....	....	....	.....
Cervicitis	....	13	....	....	9	2	1	1	.....
Cryptorchidy	....	....	....	2	1	....	....	1	.....
Dystocia	....	48	1	1	46	....	....	1	3
Examination of genitals	....	46	....	....	....	....	....	....	.....
Hematuria	....	1	....	....	....	....	....	1	.....
Hydrops, fetal membranes	....	1	....	....	....	....	....	1	.....
Intussusception, uterus	....	1	....	....	....	....	....	1	.....
Laceration, udder	....	1	....	....	....	....	....	1	.....

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Killed or died	Undetermined
5. DISEASES OF THE GENITO-URINARY SYSTEM — *Concl'd*									
Macerated fetus	....	1	1	....	1	....	....	1	.....
Mastitis	....	80	....	....	58	5	10	4	3
Metritis	....	62	1	....	57	....	....	5	1
Mummified fetus	....	2	....	....	1	....	....	1	.....
Oedema of udder	....	2	....	....	2	....	....	....	.....
Pregnancy examination	....	86	....	....	....	....	....	....	.....
Prolapse of uterus	....	6	....	1	4	....	....	3	.....
Prolapse of vagina	....	5	3	1	6	....	....	3	.....
Pyelonephritis	....	1	....	....	....	....	....	1	.....
Retained placenta	....	99	....	....	90	....	....	7	2
Rupture of prepuce	....	1	....	....	....	....	....	1	.....
Sterility	....	81	....	....	30	....	3	11	37
Teat, amputation	....	4	....	....	4	....	....	....	.....
Teat, fistula	....	1	....	....	....	....	1	....	.....
Teat, infection	....	9	....	....	9	....	....	....	.....
Teat, obstruction	....	14	....	....	9	....	3	....	2
Teat, stenosis	....	37	....	....	30	1	1	....	5
Torsion of uterus	....	7	....	....	6	....	....	1	.....
Tumor of udder	....	1	....	....	....	....	....	....	1
Tumor of vulva	....	1	....	....	....	....	....	....	1
Vaginitis	....	14	....	....	10	1	....	1	2
6. DISEASES OF THE SKIN									
Dermatitis	6	8	1	....	12	2	1	....	.....
Furunculosis	1	....	....	....	1	....	....	....	.....
Lice	2	....	....	....	....	....	....	....	.....
Mange	3	....	....	....	2	1	....	....	.....
Ringworm	....	36	....	....	36	....	....	....	.....
Urticaria	....	2	....	....	2	....	....	....	.....
7. DISEASES OF THE NERVOUS SYSTEM									
Cerebral hemorrhage	2	....	....	....	....	....	....	2	.....
Convulsions	3	....	....	....	1	....	2	....	.....
Heat stroke	1	....	....	....	....	....	....	1	.....
Meningitis	2	4	....	....	....	....	....	1	5
Paralysis	....	2	....	....	1	....	....	1	.....
Parturient paresis	....	34	....	....	34	....	....	....	.....
8. DISEASES OF THE EYE									
Keratitis	....	3	....	....	....	3	....	....	.....
Ophthalmia, periodic	5	....	....	....	2	....	....	....	3
9. EXTERNAL DISEASES									
Abscess	5	17	....	4	25	....	....	1	.....
Amputation of claw	....	3	....	....	3	....	....	....	.....
Arthritis	10	5	....	....	4	5	2	2	2
Contusions	4	7	....	....	10	....	....	1	.....
Coxitis	1	....	....	....	1	....	....	....	.....
Distorsion, shoulder	1	....	....	....	1	....	....	....	.....
Fistulous withers	2	....	....	....	....	....	....	2	.....
Foul	....	21	....	....	21	....	....	....	.....
Fracture, metatarsus	1	....	....	....	....	....	....	1	.....
Fracture, radius	2	....	....	....	....	....	....	2	.....
Fracture, tibia	2	....	....	....	....	....	....	2	.....
Fracture, ulna	1	....	....	....	....	....	....	1	.....
Hematoma	5	4	....	....	9	....	....	....	.....
Hernia	....	2	....	....	....	....	....	2	.....
Hygroma	....	4	....	....	2	2	....	....	.....
Lameness	6	2	....	....	2	2	2	....	2
Nail prick	3	4	....	....	6	....	....	1	.....
Pododermatitis	14	3	....	....	16	1	....	....	.....
Resection flexor pedis tendon	1	....	....	....	1	....	....	....	.....
Sore neck	3	....	....	....	3	....	....	....	.....
Sore shoulders	2	....	....	....	2	....	....	....	.....
Sprain	1	1	....	....	2	....	....	....	.....
Tendinitis	2	....	....	....	2	....	....	....	.....
Tendovaginitis	1	2	....	....	3	....	....	....	.....
Thrush	11	....	....	....	11	....	....	....	.....
Wounds	38	12	17	1	50	....	....	18	.....

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Killed or died	Undetermined
10. MISCELLANEOUS DISEASES									
Amputation of tail	....	....	46	....	....	....	....	....	.....
Cast	6	2	....	....	4	....	....	4	.....
Dehorning	....	97	....	....	....	....	....	....	.....
Hog cholera immunization	....	....	....	620	....	....	....	....	.....
Inspection of carcass	....	3	....	....	....	....	....	....	.....
Physical examination	....	564	....	....	....	....	....	....	.....
Poisoning, belladonna	....	1	....	....	1	....	....	....	.....
Poor condition	5	3	....	....	3	....	3	....	2
Rachitis	....	2	....	....	....	1	....	....	1
Ringing bulls	....	12	....	....	....	....	....	....	.....
Rope burn	....	1	....	....	....	....	1	....	.....
Tuberculin test	....	709	....	....	....	....	....	....	.....
Tumors	2	5	....	....	5	....	1	....	1
Vaccination, B. abortus	....	2	....	....	....	....	....	....	.....
Vaccination, hemorrhagic septicemia	....	....	10	....	....	....	....	....	.....
Vaccination, metritis	....	19	....	....	....	....	....	....	.....
	398	2,480	223	681	....	....	....	....	.....
Total	....	....	....	....	3,782	....	....	....	.....

REPORT OF THE SURGICAL AND CONSULTING CLINIC 1924–1925

J. N. FROST AND M. H. MABEY

Department of Surgery

There has been an increase in the total number of cases over the past few years. The increase has been in horses and sheep, with a slight decrease in cattle and swine. There have been many more valuable race, saddle and general purpose horses sent to the clinic for various operations than in previous years.

Twelve goats have been treated, but they have been classed and counted with the sheep.

REPORT OF THE SURGICAL AND CONSULTING CLINICS

July 1, 1924, to *June* 20, 1925

DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Killed or died	Undetermined
Abortion	2	6	....	....	6	....	....	....	2
Abscesses	4	16	....	....	....	19	1	....	.....
Abscess (udder)	....	7	1	....	6	1	....	1	.....
Actinomyces necrophorus	....	4	1	....	4	....	....	1	.....
Actinomycosis	....	2	....	....	....	1	....	1	.....
Arthritis	3	2	....	....	3	2	....	....	.....
Atresia teat	....	1	....	....	....	....	1	....	.....
Azoturia	2	....	....	....	2	....	....	....	.....
Balanitis	6	....	....	....	6	....	....	....	.....
Botryomycosis	....	....	....	1	1	....	....	....	.....
Brittle hoof	1	....	....	....	1	....	....	....	.....
Broken hoof	....	1	....	....	....	1	....	....	.....
Burn	1	....	....	....	1	....	....	....	.....
Caesarian section	....	....	3	....	1	....	....	2	.....
Calf scours	....	4	....	....	2	....	....	2	.....
Calculi in urethra	1	....	1	....	2	....	....	....	.....
Canker foot	1	....	....	....	....	1	....	....	.....
Carcinoma	1	....	....	....	....	....	....	1	.....
Castration	3	1	13	6	23	....	....	....	.....
Chloroformed	....	1	....	....	....	....	....	1	.....
Colic	2	....	....	....	2	....	....	....	.....
Contracted feet	2	....	....	....	2	....	....	....	.....
Contusions	4	7	....	....	11	....	....	....	.....
Cow pox	....	25	....	....	25	....	....	....	.....
Cryptorchid	1	1	....	2	4	....	....	....	.....
Debilitated	6	....	....	....	4	....	....	....	2
Dehorning	....	1	....	....	1	....	....	....	.....
Dermatitis	1	....	....	....	1	....	....	....	.....
Diarrhoea	1	8	....	....	7	....	....	2	.....
Diseased tooth	5	....	....	....	4	....	....	....	1
Distemper	1	....	....	....	1	....	....	....	.....
Docking	....	....	20	....	20	....	....	....	.....
Dysentery	3	....	....	....	3	....	....	....	.....
Dystocia	1	5	1	1	7	....	....	1	.....
Eczema	1	....	....	....	....	....	....	....	1
Examination	....	2	20	....	22	....	....	....	.....
Examination, pregnancy	....	63	....	....	63	....	....	....	.....
Examination, sterility	1	....	37	....	38	....	....	....	.....
Examination, teeth	12	....	....	....	12	....	....	....	.....
Exostosis	1	....	....	....	1	....	....	....	.....
Fibroma on sheath	1	....	....	....	1	....	....	....	.....
Fistula, anal	1	....	....	....	....	....	....	....	1
Fistula, salivary	1	....	....	....	1	....	....	....	.....

DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Killed or died	Undetermined
Fistula, teat	1	....	....	....	1	....	....	....	....
Fistula, withers	9	....	....	....	7	....	....	....	2
Foot rot	....	12	15	....	26	....	....	1	.....
Fracture of external angle ilium	....	1	....	....	1	....	....	....	.....
Glanders test	4	....	....	....	4	....	....	....	.....
Gonitis	2	2	....	....	3	....	....	1	.....
Hair root infection	5	....	....	....	5	....	....	....	.....
Heat stroke	1	....	....	....	1	....	....	....	.....
Heaves	7	....	....	....	....	3	....	....	4
Hematoma	3	6	....	....	9	....	....	....	.....
Hemorrhage, horn	....	1	....	....	1	....	....	....	.....
Hernia, scrotal	1	....	....	9	10	....	....	....	.....
Hernia, umbilical	2	....	....	....	1	....	....	1	.....
Hernia, ventral	....	....	1	....	....	....	1	....	.....
Horn tumor	1	....	....	....	....	1	....	....	.....
Hydrothorax	1	....	....	....	....	....	....	1	.....
Hygroma	2	....	....	....	2	....	....	....	.....
Impaction	1	....	....	....	1	....	....	....	.....
Impaction teat	....	2	....	....	2	....	....	....	.....
Indigestion	15	10	....	....	23	....	....	2	.....
Infection hock	2	....	....	....	1	....	....	1	.....
Inflammation of cheek ligament	2	....	....	....	2	....	....	....	.....
Influenza	2	....	....	....	2	....	....	....	.....
Injury	6	2	....	....	6	2	....	....	.....
Injury to foot	2	....	....	....	2	....	....	....	.....
Intestinal catarrh	2	....	....	....	2	....	....	....	.....
Keratitis	....	....	10	....	8	2	....	....	.....
Lameness	10	....	....	....	8	1	1	....	.....
Laminitis	3	....	....	....	3	....	....	....	.....
Laryngitis	1	....	....	....	1	....	....	....	.....
Lice	3	....	....	....	3	....	....	....	.....
Lymphangitis	3	....	....	....	3	....	....	....	.....
Macerated fetus	....	....	1	....	....	....	....	1	.....
Mange, psoroptic	....	....	2	....	1	....	....	....	1
Mastitis	....	21	....	....	17	3	1	....	.....
Meconium retained	3	1	....	....	3	....	....	1	.....
Meningitis	....	1	....	....	....	....	....	1	.....
Metritis	6	26	....	....	26	2	4	....	.....
Myotomy	2	....	....	....	2	....	....	....	.....
Navel infection	....	2	....	....	2	....	....	....	.....
Nymphomania	4	....	....	....	4	....	....	....	.....
Open joint	3	....	....	....	1	....	....	2	.....
Ophthalmia, periodic	5	....	....	....	....	5	....	....	.....
Otitis media	....	2	....	....	1	1	....	....	.....
Paralysis	1	....	11	....	2	....	....	10	.....
Paralysis, radial	2	....	....	....	2	....	....	....	.....
Parasites	1	....	300	....	1	....	....	....	300
Parturient paresis	....	2	....	....	2	....	....	....	.....
Pericarditis, traumatic	....	1	....	....	....	....	....	1	.....
Pharyngitis	....	....	1	....	1	....	....	....	.....
Placenta, retained	3	17	....	....	20	....	....	....	.....
Pneumonia	1	2	....	....	2	....	....	1	.....
Pododermatitis	3	2	11	....	14	2	....	....	.....
Poll evil	4	....	....	....	3	1	....	....	.....
Prolapse, rectum	....	....	1	6	6	....	....	1	.....
Prolapse, uterus	....	1	....	....	1	....	....	....	.....
Prolapse, vagina	....	....	1	....	1	....	....	....	.....
Quarter crack	1	....	....	....	....	1	....	....	.....
Retention cyst of parotid duct	....	1	....	....	....	....	....	....	1
Ring bone	2	....	....	....	1	....	1	....	.....
Ring worm	....	5	....	....	5	....	....	....	.....
Ringing bull	....	1	....	....	1	....	....	....	.....
Roarer	5	....	....	....	4	....	1	....	.....
Rumenotomy	....	2	....	....	2	....	....	....	.....
Sclerostoma	12	....	....	....	6	....	....	1	5
Scours	....	4	....	....	3	....	....	1	.....
Septicemia	....	....	3	....	....	....	....	3	.....
Septicemia hem. bacterin treatment	....	....	60	....	60	....	....	....	.....
Shear mouth	2	....	....	....	....	2	....	....	.....
Shoulder abscess	1	....	....	....	1	....	....	....	.....
Shoulder tumor	3	....	....	....	3	....	....	....	.....

DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Killed or died	Undetermined
Sitfast	5	....	....	....	5	....	....	....	.....
Spavin	2	....	....	....	2	....	....	....	.....
Stenosis teat	....	4	....	....	4	....	....	....	.....
Sterility	4	10	....	....	10	....	2	2	.....
Strangles	5	....	....	....	5	....	....	....	.....
Stronglidae	11	....	....	....	9	....	2	....	.....
Teeth, diseased	11	....	....	....	....	11	....	....	.....
Teeth, sharp	51	....	....	....	51	....	....	....	.....
Teeth, split	8	....	....	....	8	....	....	....	.....
Tendinitis	5	4	....	....	6	3	....	....	.....
Tendo-vaginitis	....	1	....	....	....	1	....	....	.....
Tetanus	2	....	....	....	....	....	....	2	.....
Thrush	2	....	....	....	....	2	....	....	.....
Tracheitis	....	....	1	....	....	....	....	1	.....
Traumatic gastritis	....	3	....	....	....	3	....	....	.....
Trimming claws	....	6	....	....	6	....	....	....	.....
Trochanteric lameness	1	....	....	....	....	1	....	....	.....
Tuberculin test	....	150	....	....	147	....	....	3	.....
Tuberculosis	....	1	....	....	....	....	....	1	.....
Tumor, ear	1	....	....	....	1	....	....	....	.....
Tumor, shoulder	5	....	....	....	5	....	....	....	.....
Urachus, pervious	1	....	....	....	1	....	....	....	.....
Urticaria	....	2	....	....	2	....	....	....	.....
Vaginitis	....	1	....	....	1	....	....	....	.....
Wooden tongue	....	1	....	....	1	....	....	....	.....
Wounds, calk	14	....	....	....	14	....	....	....	.....
Wounds, castration	....	....	8	....	7	....	....	1	.....
Wounds, coronary band	1	....	....	....	1	....	....	....	.....
Wounds, kick	11	....	....	....	9	....	....	2	.....
Wounds, miscellaneous	13	....	....	....	13	....	....	....	.....
Wounds, nail	3	3	....	....	6	....	....	....	.....
Wounds, stub	2	....	....	....	2	....	....	....	.....

Horses	383
Cattle	470
Sheep	523
Swine	25
	1,401
Counted twice	30
Total	1,371

REPORT OF THE SMALL ANIMAL CLINIC

July 1, 1924, *to June* 30, 1925

H. J. MILKS and H. C. STEPHENSON

Department of Materia Medica and Small Animal Clinic

The variety of cases treated in the Small Animal Clinic was much the same as in previous years. In actual number there was an increase of 243 cases, bringing the total number up to 1,638. There has been as much material as could be used in our clinical instruction and opportunity offered to stress aseptic and antiseptic surgery, local and general anesthesia, the modern treatment of wounds and the principles of treatment of internal diseases.

As a whole, our results have been satisfactory. The mortality is comparatively high but it is easily explained by the number of serious and moribund cases that are brought to us from other localities for consultation.

CASES TREATED IN THE SMALL ANIMAL CLINIC

NAME OF DISEASE	Dogs	Cats	Recovered	Improved	Not improved	Died	Destroyed	Undetermined
1. INFECTIOUS DISEASES								
Distemper	138	....	74	....	....	52	10	2
Infectious gastro-enteritis	....	39	7	....	....	32	....	.....
Infectious hemorrhagic gastro-enteritis	9	....	1	....	....	8	....	.....
2. DISEASES OF THE CIRCULATORY SYSTEM								
Dirofilaria immitis	1	....	....	1	....	....	....	.....
3. DISEASES OF THE RESPIRATORY SYSTEM								
Bronchitis	8	9	8	4	1	2	1	1
Coryza	6	9	15	....	....	....	....	.....
Laryngitis	4	2	6	....	....	....	....	.....
Pleuritis	1	....	....	....	....	1	....	.....
Pneumonia	1	1	....	....	....	2	....	.....
Sinusitis	4	2	3	1	....	1	....	1
4. DISEASES OF THE NERVOUS SYSTEM								
Chorea	4	....	....	....	3	....	1	.....
Convulsions	15	4	12	2	....	2	1	2
Encephalitis	3	....	....	....	....	2	1	.....
Hysteria	1	....	1	....	....	....	....	.....
Paralysis, posterior	4	....	1	....	....	2	1	.....
Paralysis, radial	1	....	....	....	1	....	....	.....
Paralysis, vestibular	1	1	1	....	....	1	....	.....
5. DISEASES OF THE EYE								
Amaurosis	2	....	....	1	1	....	....	.....
Conjunctivitis, catarrhal	15	2	14	2	1	....	....	.....
Conjunctivitis, follicular	2	....	2	....	....	....	....	.....
Eczema of eyelids	1	....	1	....	....	....	....	.....
Entropion	6	....	6	....	....	....	....	.....
Eyeball, congenital absence of	1	....	....	....	....	....	1	.....
Eyeball, enucleation of	2	....	2	....	....	....	....	.....
Foreign body in eye	1	1	2	....	....	....	....	.....
Hydropthalmos	1	....	....	....	1	....	....	.....

NAME OF DISEASE	Dogs	Cats	Recovered	Improved	Not improved	Died	Destroyed	Undetermined
Iritis	1	....	1	....	....	....	....	.....
Keratitis, superficial	5	....	5	....	....	....	....	.....
Keratitis, ulcerative	12	2	14	....	....	....	....	.....
Membrana nictitans, removal of protruding	2	....	2	....	....	....	....	.....
Orbital gland, removal of tumefied	6	....	6	....	....	....	....	.....
Staphyloma	....	1	1	....	....	....	....	.....
6. DISEASES OF THE GENITO-URINARY SYSTEM								
Balanitis	2	....	2	....	....	....	....	.....
Caesarian section	6	....	6	....	....	....	....	.....
Calculi	2	....	1	....	....	....	1	.....
Castration	8	57	61	....	....	4	....	.....
Cystitis, hemorrhagic	1	....	1	....	....	....	....	.....
Dystocia	1	1	2	....	....	....	....	.....
Eclampsia	1	....	1	....	....	....	....	.....
Incontinence	2	....	....	2	....	....	....	.....
Mastitis	1	....	1	....	....	....	....	.....
Metritis	4	....	3	....	....	1	....	.....
Nephritis	....	1	....	....	....	1	....	.....
Oestrum	4	....	....	....	....	....	....	.....
Oophorectomy	263	114	372	....	....	5	....	.....
Paraphymosis	1	....	1	....	....	....	....	.....
Parturition	4	1	5	....	....	....	....	.....
Penis, congenital malformation of	1	....	1	....	....	....	....	.....
Prostatitis	2	....	....	....	....	1	....	1
Sterility	1	....	....	....	....	....	1	.....
Urethra, stricture of	1	....	....	1	....	....	....	.....
Vagina, douching of	2	....	....	....	....	....	....	.....
Vagina, eversion of	1	....	1	....	....	....	....	.....
Vagina, stricture of	1	....	....	....	1	....	....	.....
7. DISEASES OF THE DIGESTIVE SYSTEM								
Constipation	3	1	4	....	....	....	....	.....
Constipation, false	1	....	1	....	....	....	....	.....
Diarrhea	....	4	4	....	....	....	....	.....
Foreign body in mouth	1	....	1	....	....	....	....	.....
Foreign body in pharynx	1	....	1	....	....	....	....	.....
Gastritis	27	3	28	....	....	2	....	.....
Gastro-enteritis	6	....	....	....	....	6	....	.....
Icterus	1	....	....	....	....	1	....	.....
Impaction of crop	....	*1	1	....	....	....	....	.....
Indigestion	13	†3	16	....	....	....	....	.....
Obstruction of small intestines	3	1	....	....	....	4	....	.....
Oesophagus, dilatation of	1	....	....	....	....	1	....	.....
Pharyngitis	2	....	2	....	....	....	....	.....
Ptyalism	1	....	....	....	1	....	....	.....
Rectum, paresis of	1	1	....	....	....	....	1	1
Rectum, prolapse of	....	2	2	....	....	....	....	.....
Salivary duct, obstruction of	1	....	1	....	....	....	....	.....
Stomach, acute dilatation of	2	....	1	....	....	1	....	.....
Worms, hook	5	....	2	1	1	....	....	1
Worms, round	25	7	30	....	....	2	....	.....
Worms, tape	11	1	11	1	....	....	....	.....
8. DISEASES OF THE SKIN								
Acanthosis	2	....	....	2	....	....	....	.....
Eczema	58	....	54	4	....	....	....	.....
Fleas	35	6	....	....	....	....	....	.....
Furunculosis	3	....	3	....	....	....	....	.....
Lice	3	....	....	....	....	....	....	.....
Mange, follicular	79	....	42	7	2	3	19	6
Mange, otodectic	27	8	33	2	....	....	....	.....
Mange, sarcoptic	16	‡27	30	2	1	6	4	.....
Pruritis	1	....	1	....	....	....	....	.....
Ringworm	12	....	12	....	....	....	....	.....
Seborrhea	1	....	....	....	1	....	....	.....
Urticaria	1	....	1	....	....	....	....	.....

* 1 turkey. † 1 rabbit ‡ 1 ferret

NAME OF DISEASE	Dogs	Cats	Recovered	Improved	Not improved	Died	Destroyed	Undetermined
9. EXTERNAL DISEASES								
Abscess	19	‡11	29	....	....	1	....	.....
Amputation of dewclaws	12	....	12	....	....	....	....	.....
Amputation of digit	1	....	1	....	....	....	....	.....
Amputation of tail	24	2	26	....	....	....	....	.....
Anal sacs, impaction of	8	....	8	....	....	....	....	.....
Ankylosis of elbow	1	....	....	....	1	....	....	.....
Ascites	1	....	....	....	1	....	....	.....
Carcinoma	5	3	2	....	....	3	2	1
Craniotomy	1	....	1	....	....	....	....	.....
Cuterebra	....	4	4	....	....	....	....	.....
Fracture of femur	9	2	10	....	....	....	1	.....
Fracture of femur, compound	1	....	1	....	....	....	....	.....
Fracture of fibula	1	....	1	....	....	....	....	.....
Fracture of mandible, compound	1	1	1	....	....	....	1	.....
Fracture of metacarpi, compound	1	2	3	....	....	....	....	.....
Fracture of metatarsi, compound	....	2	1	....	....	....	1	.....
Fracture of pelvis	3	....	2	....	....	....	1	.....
Fracture of radius and ulna	4	....	4	....	....	....	....	.....
Fracture of radius and ulna, compound	1	....	....	....	....	....	1	.....
Fracture of ribs	1	....	1	....	....	....	....	.....
Fracture of scapula	1	....	1	....	....	....	....	.....
Fracture of spine	1	....	....	....	....	....	1	.....
Fracture of sternum	1	....	1	....	....	....	....	.....
Fracture of tibia and fibula	3	1	3	....	....	....	1	.....
Fracture of tibia and fibula, compound	2	2	....	....	....	....	4	.....
Fracture of ulna	1	....	1	....	....	....	....	.....
Harelip	1	....	1	....	....	....	....	.....
Hernia, inguinal	1	....	1	....	....	....	....	.....
Hernia, pseudoperineal	1	....	....	1	....	....	....	.....
Hernia, umbilical	9	....	9	....	....	....	....	.....
Hernia, ventral	1	1	1	1	....	....	....	.....
Injuries, internal	9	1	5	....	....	4	1	.....
Lameness	12	....	12	....	....	....	....	.....
Luxation of elbow	1	....	1	....	....	....	....	.....
Luxation of hip	5	....	3	2	....	....	....	.....
Luxation of patella	3	....	1	....	2	....	....	.....
Luxation of shoulder	1	....	1	....	....	....	....	.....
Needle in neck	....	1	1	....	....	....	....	.....
Otorrhea, catarrhal	15	....	12	2	....	....	1	.....
Papilloma	7	....	7	....	....	....	....	.....
Peritonitis	2	....	1	....	....	1	....	.....
Sarcoma	1	....	....	....	....	....	1	.....
Scald	3	1	4	....	....	....	....	.....
Spine, contusion of	1	....	1	....	....	....	....	.....
Stifle, open joint	....	1	....	....	....	....	1	.....
Teeth, cleaning	1	2	3	....	....	....	....	.....
Teeth, extraction of	2	4	6	....	....	....	....	.....
Torticollis	1	1	....	....	....	2	....	.....
Tumefied ear flap	1	2	3	....	....	....	....	.....
Tumors, unclassified	1	*1	2	....	....	....	....	.....
Wounds	48	16	58	....	1	4	1	.....
10. MISCELLANEOUS								
Autopsy	2	1	....	....	....	....	....	.....
Bumblefoot	....	*1	1	....	....	....	....	.....
Destroy	41	87	....	....	....	....	....	.....
Examination	12	....	....	....	....	....	....	.....
Goiter	4	....	1	1	1	....	....	1
Observation	3	....	....	....	....	....	....	.....
Poisoning, roach	1	....	1	....	....	....	....	.....
Rheumatism	13	....	6	7	....	....	....	.....
Rickets	4	1	4	1	....	....	....	.....
Suspected rabies	3	....	....	....	....	....	....	.....
Undetermined	5	2	....	....	....	....	....	.....
Vaccination, distemper	6	....	....	....	....	....	....	.....
	1,234	465						

*1 turkey

Total cases	1,699
Counted twice	61
Net total	1,638

REPORT OF THE DIAGNOSTIC WORK ON POULTRY DISEASES

July 1, 1924 *to June* 30, 1925

E. L. BRUNETT and J. M. HENDRICKSON
Department of Pathology and Bacteriology

The specimens received during the past fiscal year show a large increase over those received during the preceding year. A total of 646 accessions or cases were recorded. This as compared with the 413 accessions of the preceding year represents a 56 per cent increase in this particular phase of the poultry work.

The material actually autopsied consisted of 1,314 specimens. These were divided into 1,265 chickens, 20 turkeys, 14 ducks, 2 pigeons, 5 grouse, 2 canaries and 6 crows.

The mass of correspondence concerning flock troubles of a disease nature is not recorded in this report. The general interest of the poultry people is indicated by the yearly increase in the amount of diagnostic work. The need for a better understanding of the various diseases of poultry becomes more pronounced as the poultry industry expands. It is becoming apparent that the poultrymen no longer accept the normal mortality without question. They are becoming more alert to inquire into the early losses than to wait until the entire flock has been depleted.

BACILLARY WHITE DIARRHEA

During the past year a large number of chicks received at the laboratory from outbreaks associated with a high mortality revealed that they had died of *Bact. pullorum* infection. The total number of accessions does not bring out the enormous number of chicks which die annually of this particular disease in this State. If it were possible to accurately record all of the outbreaks, the seriousness of this disease would be better realized.

The disease is widespread, not only in this State, but in others as well. A number of outbreaks were traced back to hatcheries outside of the State. The exposure and improper care associated with long trips, no doubt, play an important part in the mortality produced by this disease in shipped chicks. The infection must necessarily be present in the beginning, but any factors which tend to lower the resisting forces of the chick's body aid considerably in allowing an outbreak to occur, resulting in a high mortality.

The breeders in 22 flocks were tested for *Bact. pullorum* infection. The certified stock was tested in 17 of the above flocks. Two hundred and eighty-six, or .09 per cent out of 2,954 certified females, reacted to the agglutination test. Ten, or .02 per cent out of 473 certified males, reacted to the same test. The percentage of reactors in the different certified flocks varied among the females from 0 to 35 per cent, in the certified males from 0 to 16 per cent.

In the majority of the certified flocks there had not been any testing done previous to this year. The owners were not aware, with one or two exceptions, that the disease existed among the offspring. The dormancy of the infection should not be allowed to deceive the poultry owners into believing that their flocks are entirely free from the infection.

The para-typhoid infection of poultry is a problem which has presented many difficulties and unsolved questions which are demanding more attention each year. With the accumulation of information concerning the cycles of infection, it is hoped that this economically important group of diseases may be more successfully controlled.

The necrotic foci observed in the gizzard musculature, lungs and heart, as referred to in the 1923–24 report, were found quite frequently during the past year. The fact that they were observed in chicks ten days old, and older, indicates a chronic form of the disease. The low virulence of the invading organisms or a late infection with an associated increased resistance of the body tissues, is the probable explanation.

In three outbreaks of a fatal septicemia among chicks an organism indistinguishable from *Bact. sanguinarium* (fowl typhoid) was isolated.

OTHER ACUTE INFECTIONS

It cannot be said that fowl cholera has caused a considerable amount of trouble in the flocks during the past year. The losses caused by this disease, however, are not to be ignored. In a number of the outbreaks the losses have totaled as high as 30 per cent of the flock. The tendency of the disease to become chronic and later acute was very apparent in some of the outbreaks. The use of autogenous bacterins and the application of sanitary measures about the premises have been successful in controlling outbreaks in this State..

The differentiation of fowl cholera from other acute infections is important. Apoplectiform septicemia (streptococcus infection) is an acute disease which may become confused with fowl cholera. The short course, sudden appearance and termination will serve to differentiate it somewhat. A bacteriological examination is, however, necessary for an accurate diagnosis. Outbreaks of the disease in this State have responded to the application of sanitary measures.

The occurrence of fowl typhoid has not been very frequent if we are able to judge by the specimens received.

The para-typhoid infections refer to an outbreak of "keel" in ducklings caused by *Bact. anatum,* the disease being somewhat analogous to bacillary white diarrhea.

An outbreak of an acute disease among canaries in a large "Pet Shop" furnished an organism identified as *B. pestiscaviae.*

Fowl plague, which is discussed elsewhere, caused considerable havoc both in Greater New York and in some of the upstate markets and farms. It is estimated that between 500,000 and 600,000

fowls died of the disease in Greater New York. These figures do not include those dying or killed in other market terminals. The losses on the farms and upstate markets of this State can be placed at a conservative estimate of 60,000.

TUBERCULOSIS

The apparent prevalence of this disease in certain sections of the State makes it an important one. It is a disease usually occurring in a chronic form and hence may gain headway in a flock before its presence is recognized. In the sections where the disease has gained a foothold, it is being thoughtlessly disseminated by the selling of infected birds. The early recognition of this disease is important so that eradication measures can be applied. The use of the intradermal tuberculin test, followed by a proper disinfection of the houses, furnishes a practical means for the eradication of the disease in flocks with a low percentage of infected fowls. The disposal of the entire flock and the restocking with healthy fowls, after thoroughly disinfecting the premises, is the best procedure, where there is evidence of a high percentage of infection.

The remainder of the report on the diagnostic work needs no special comment at this time. Exception may be taken to the two outbreaks listed under edema of the wattles. The fact that this condition is repeatedly mistaken for injury to the wattles, frozen wattles, etc., perhaps does not give us an accurate idea of its occurrence. The condition appeared both preceding and following outbreaks of fowl cholera. Both hens and cockerels were affected.

The wattles become swollen and are soft and pliable during the early stages. The formation of purulent material between the folds of skin forming the wattles soon becomes apparent. Upon incision of the infected wattle, in the later stages of the disease, a cheesy, purulent material is found present. Bacteriological examination of this material revealed in the limited number of cases examined, *P. avisepticus.*

The inoculation of the purulent material from these cases, or cultures of *P. avisepticus* isolated from them, results in a reproduction of the condition. Cropping the wattles has been recommended in the states where this particular affection is prevalent.

It is important that this condition be recognized as its possible influence in the control of fowl cholera is not to be ignored.

DIAGNOSES

DISEASE	CHICKENS		Turkeys	Ducks	Pigeons	Grouse	Canaries	Crows
	Young	Adult						
Specific bacterial diseases								
Bacillary white diarrhea								
Bact. pullorum infection	88	3	....	....	....	....	....	.....
Bact. sanguinarium infection	3	....	....	....	....	....	....	.....
Fowl cholera	....	13	....	....	....	....	....	.....
Fowl typhoid	....	1	....	....	....	....	....	.....
Para-typhoid septicemia	....	....	....	1	....	....	1	.....
Streptococcus infection	2	5	....	....	....	....	....	.....
Tuberculosis	....	18	....	....	....	....	....	.....
Filterable virus diseases								
Fowl plague	....	22	1	....	....	....	....	.....
General bacterial diseases								
Abscess	....	1	....	....	....	....	....	.....
Bumble foot	....	1	....	....	....	....	....	.....
Edema of wattles	....	2	....	....	....	....	....	.....
Pericarditis	....	1	....	....	....	....	....	.....
Peritonitis	....	6	....	....	....	....	....	.....
Pneumonia	....	3	....	....	....	....	....	.....
Subcutaneous infection	....	1	....	....	....	....	....	.....
General diseases								
Bile duct occlusion	....	2	....	....	....	....	....	.....
Blindness	....	4	....	....	....	....	....	.....
Cardiac rupture	....	2	....	....	....	....	....	.....
Cecal ulcers	....	1	....	....	....	....	....	.....
Edema, subcutaneous	....	1	....	....	....	....	....	.....
Enteritis, catarrhal	....	2	....	....	....	....	....	.....
Enteritis, hemorrhagic	....	2	....	....	....	....	....	.....
Foot necrosis	....	....	....	1	....	....	....	.....
Frozen comb	....	1	....	....	....	....	....	.....
Hemorrhage, external	....	4	....	....	....	....	....	.....
Hemorrhage, internal	....	8	....	....	....	....	....	.....
Hemorrhage, ovarian	....	1	....	....	....	....	....	.....
Hemorrhagic cloacitis	....	1	....	....	....	....	....	.....
Impaction, duodenum	....	1	....	....	....	....	....	.....
Impaction, gizzard	1	....	....	....	....	....	....	.....
Impaction, oviduct	....	14	....	....	....	....	....	.....
Internal layer	....	11	....	....	....	....	....	.....
Leukemia and pseudo-leukemia	....	8	....	....	....	....	....	.....
Obesity	....	2	....	....	....	....	....	.....
Poisoning, phosphorus	....	1	....	....	....	....	....	.....
Prolapse oviduct	....	3	....	....	....	....	....	.....
Tumor	....	18	....	....	....	....	....	.....
Unabsorbed yolk sac	1	....	....	....	....	....	....	.....
Diseases of the head								
Avian diphtheria	....	22	....	....	....	....	....	.....
Chicken pox	....	5	1	....	1	....	....	.....
Roup	....	3	....	....	....	....	....	.....
Roup with canker formation	....	4	....	....	....	....	....	.....
Nutritional diseases								
Nutritional disease with esophageal pustules	....	6	....	....	....	....	....	.....
Nutritional disease with nephritis alone	4	21	....	....	....	....	....	....
Visceral gout	....	18	....	....	....	....	....	.....
Parasites								
Lice	....	6	1	....	....	....	....	.....
Filaria, blood	....	....	....	....	....	....	....	1
Mites								
Cytodites nudus (air sac mite)	....	3	....	....	....	....	....	.....
Dermanysus gallinae (red mite)	....	1	....	....	....	....	....	.....
Roundworms								
Capillaria (small intestinal)	1	24	....	....	....	....	....	.....
H. perspicillum (large intestinal)	2	25	....	....	....	....	....	.....
H. vesicularis (cecal)	2	11	....	....	....	2	....	.....
Tapeworms	....	46	....	1	....	....	....	.....
Protozoan diseases								
Coccidiosis	70	9	5	....	....	....	....	.....
Entero-hepatitis	7	....	8	....	....	1	....	.....
Fungus diseases								
Aspergillosis	1	....	....	....	....	....	....	.....

DISEASE	CHICKENS		Turkeys	Ducks	Pigeons	Grouse	Canaries	Crows
	Young	Adult						
Miscellaneous								
Decomposed	17	6	....	1	....	....	....	.....
Negative examination	17	13	....	....	....	....	....	.....
Normal	1	2	....	....	....	....	....	.....
Unclassified	....	78	....	....	....	....	....	.....
	217	467	16	4	1	3	1	1
Counted twice	7	57						
	210	410	16	4	1	3	1	1

REPORT OF THE DIAGNOSIS LABORATORY

FOR THE YEAR ENDING JUNE 30, 1925

C. M. CARPENTER
Department of Pathology and Bacteriology

The diagnostic work is now being carried on in the south wing, first floor, of James Law Hall. The new laboratory is well equipped for making bacteriological, pathological and serological examinations upon diseases of animals. As stated in the last report, the diagnostic work upon the diseases of birds is in charge of Dr. E. L. Brunett. Besides the regular routine work, research has been done on "Skin Tuberculosis of Cattle," which is appended to this report. Much time has been spent on studies of *Brucella abortus* infection in cattle. One case of this infection has occurred in a student attending the University and considerable time has been spent in studying this case and attempting to trace the source of infection as well as the biological characteristics of the strain isolated from the blood and urine of the patient. This has necessitated the examination of many samples of milk and the use of a considerable number of experimental animals. Studies on a mixed infection with *Brucella abortus* and *Mycobacterium tuberculosis* in guinea pigs have been carried out by A. F. Schoenfeld and the writer. It was found that these two diseases can be produced in guinea pigs simultaneously and that they can readily be identified should tissues or milk containing both organisms be present in the sample injected. The acid fast tubercle bacillus is demonstrated as usual in smears made from the nodules in the spleen, liver, lymph glands, lungs, etc., while *Brucella abortus* may be recovered in pure culture upon any medium suitable for its cultivation. The medium suitable for growing *Brucella abortus* fails to develop the growth of *Mycobacterium tuberculosis.*

The specific agglutinins for *Brucella abortus* are present in the blood sera of guinea pigs suffering from both infections. It is somewhat difficult to differentiate macroscopically between the lesions of tuberculosis and abortion disease. However, in the male guinea pigs *Brucella abortus* produces abscesses in the epididymes and atrophy of the testes. These lesions are seldom produced in guinea pigs by the tubercle bacillus.

RABIES

On March 4, 1925, a dog's head was received at the laboratory, the brain of which showed the presence of Negri bodies. This was the first positive case of rabies that had come to the laboratory since September 7, 1923. Since receiving the positive case mentioned above, we have examined seven other cases that have shown the presence of Negri bodies, making a total of 8 cases. During the fiscal year 60 examinations for Negri bodies were made. They

were distributed as follows: Dog, 55; cat, 2; cow, 1; horse, 1; squirrel, 1. The positive cases were received from Long Island, Westchester county and Orange county and one from Rochester, N. Y.

ANTHRAX, BLACKLEG, HEMORRHAGIC SEPTICAEMIA

We have received 29 specimens to be examined for anthrax and have found three cases of the disease. Last year not a single positive case was diagnosed. We have not diagnosed any cases of blackleg, but several cases of hemorrhagic septicaemia in sheep and swine have occurred. (See Table I.)

GLANDERS

Eight samples of blood serum have been forwarded to us to be tested for the presence of agglutinins. These were sent to the Department of Health in New York City for the agglutination test, as was done last year. Five of the samples showed a titre of over 1-500 while the other three were negative.

MASTITIS

One of the great problems confronting the practitioner for treatment is mastitis. One hundred twenty-six samples of mastitis milk or inflammatory exudates from infected udders have been received and autogenous bacterins and living suspensions have been prepared from cultures isolated from the majority of these samples. Very favorable reports on the use of these in the treatment of mastitis have been made to the laboratory. As has been reported before, we have isolated the alpha type of the streptococcus in the majority of the cases, this type having been isolated from 100 out of the 126 samples.

DISEASES OF THE SILVER BLACK FOX

Quite a large number of samples of feces of foxes have been received at the laboratory to be examined for parasites or their ova. These have shown for the most part hookworms and ascaris eggs. Several carcasses have been received and post mortem examinations made upon them. Extensive otorrhea was present in some of the cases, though usually there was found an acute pneumonia and enteritis. These have been called forms of fox distemper, or fox influenza. The condition is similar to distemper in dogs. Studies have been made on the bacteriology and pathology of the lesions found in the tissues of the foxes which were posted, but the work has not progressed far enough to warrant a report at this time.

DISEASES OF THE REPRODUCTIVE ORGANS OF CATTLE

It will be noted in Table I that there has been a marked increase in the number of agglutination tests made for *Brucella abortus*

infection in cattle. During the year 1923–24, 491 tests were made, while during the year 1924–25, 1,542 samples were examined. In certain herds blood samples have been collected at comparatively regular intervals and tested in an attempt to control the disease in the herds by eliminating the reactors. In one herd the reactors to the agglutination test have not been removed and the spread of the disease has been studied. Many milk samples from the herds from which blood tests have been made have been injected into guinea pigs and cultured for the presence of *Brucella abortus*. A report of these results will be made later.

Various other tissues, such as fetuses, placentas, and genital tracts, have been examined bacteriologically. Examinations of the reproductive organs of several bulls that showed the presence of agglutinins in their blood sera have been completed. *Brucella abortus* was found either in the epididymes or seminal vesicles of these animals.

Samples of semen are being sent to the laboratory more frequently than heretofore. A total of 29 samples have been examined. *Brucella abortus* was isolated by guinea pig injection from a sample of semen collected from a bull that showed the presence of *Brucella abortus* agglutinins. The sample of semen was collected by massaging the seminal vesicles per rectum. This is the first time in our experience that the Bang organism has been recovered from a sample of semen.

BIOLOGICS PREPARED AND DISTRIBUTED BY THE DIAGNOSTIC LABORATORY

During the year 37,230 mils of tuberculin have been prepared and sent out by the laboratory (See Table II); 29,712 mils of this amount were subcutaneous, while 3,742 mils and 3,776 mils were intradermal and opthalmic respectively. The total amount represents 52,446 doses of tuberculin. This is 20,986 doses less than was distributed during the year 1923–24. A total of 21,850 mils of autogenous bacterins or suspensions of living organisms were prepared, which is an increase of 5,740 mils over the amount prepared and distributed during the previous year.

Because of increased refrigerator space and shipping facilities, the diagnostic laboratory has taken over the shipping of anti-hog cholera serum and hog cholera virus, which is prepared at the Experiment Station. The amounts of these two products are tabulated elsewhere in this report.

TABLE I

Examinations Made by Diagnostic Laboratory During the Year ending June 30, 1925

MORBID CONDITIONS	Positive	Negative
Abortion, bovine (due to *Brucella abortus*)		
Agglutination tests, blood sera	402	1140
Bacteriological, fetuses	6	....
milk	16	....
placentas	8	....
uterine discharges	9	....
Abortion, bovine (*Brucella abortus* not isolated)		
Bacteriological, fetuses	6	....
milk	33	....
placentas	11	....
uterine discharges	17	....
Abscesses, bovine		
B. pyogenes	5	....
Streptococcus	3	....
Undetermined	3	....
Equine		
Staphylococcus	2	....
Actinomycosis, bovine	6	....
Anthrax	3	26
Bacterins prepared		
Mastitis	....	....
Miscellaneous	....	....
Blackleg, bovine	....	1
Botulism, equine	4	....
Broncho-pneumonia		
Calf	3	....
Cow	3	....
Dog	2	....
Fox	7	....
Sheep	3	....
Swine	3	....
Coccidiosis		
Dog	1	....
Fox	1	....
Rabbit	1	....
Fat necrosis	3	....
Feces examinations		
Cat	....	1
Belascaris	1	....
Dog	....	42
Belascaris	9	....
Hookworms	15	....
Tapeworms	2	6
Toxascaris	10	....
Whipworms	11	....
Fox		
Belascaris	2	....
Hookworm	2	....
Horse		
Ascaris	7	5
Strongylus	16	....
Sheep		
Stomach worms	1	....
Glanders		
Agglutination tests	5	3

MORBID CONDITIONS	Positive	Negative
Hemorrhagic septicaemia		
Bovine	....	10
Ovine	13	4
Porcine	3	1
Hog cholera	12	2
Johne's disease	5	5
Mastitis, bovine		
Alpha hemolytic streptococcus	100	....
Beta hemolytic streptococcus	5	....
B. coli	8	....
Staphylococcus aureus	2	....
Mixed and undetermined	11	....
Mange		
Bovine	....	14
Follicular	1	....
Sarcoptic	6	....
Canine	....	6
Follicular	1	....
Sarcoptic	4	....
Equine	....	6
Fox		
Otodectic	4	....
Ovine	....	1
Psoroptic	2	....
Necrobacillosis		
Bovine	10	....
Ovine	1	....
Parasitic diseases (See mange and feces examinations)		
Ascariasis		
Canine	1	....
Fox	2	....
Lung worms		
Swine	3	....
Nodular disease		
Ovine	5	....
Stomach worms		
Ovine	1	....
Tapeworms		
Canine	1	....
Equine	1	....
Feline	2	....
Ovine	1	....
Rabies		
Bovine	....	1
Canine	8	47
Equine	....	1
Feline	....	2
Squirrel	....	1
Semen (bacteriological and microscopic examinations)	29	....
Sterility, bovine		
Chronic productive salpingitis	8	....
Metritis due to *B. pyogenes*	1	....
Undetermined	1	....
Tetanus		
Ovine	2	....
Tuberculosis		
Bovine		
Milk	1	15
Pus	2	6
Skin	25	1
Sputum	....	1
Tissue other than skin	11	13
Porcine		
Tissue	....	2

Morbid Conditions		
Tumors	Positive	Negative
Carcinoma		
Bovine	4	....
Papilloma		
Bovine	1	....
Canine	2	....
Tissues, unsatisfactory condition when received	9	....
Miscellaneous examinations	110	....
Unable to make diagnosis	24	....
	1085	1363
Total	2448	
Counted twice	32	
Number of examinations	2416	

TABLE II

Biologies Prepared by the Diagnostic Laboratory and Sent Out During the Year Ending June 30, 1924

Tuberculin—subcutaneous (mils)	29,712
intradermal (mils)	3,742
ophthalmic (mils)	3,776
Total (mils)	37,230
(The above amounts of the various kinds of tuberculin represent 52,446 doses.)	
Mallein (mils)	10
Total (mils)	10
Autogenous vaccines and bacterins	
Hemorrhagic septicaemia (mils)	1,600
Mastitis bacterins	13,280
Miscellaneous bacterin (mils)	6,970
	21,850

AUTOPSIES

S. A. GOLDBERG and R. H. VOLGENAU
Department of Pathology and Bacteriology

We had an unusually large number of autopsies this year. This is accounted for by the heavy mortality from canine distemper and feline enteritis. As has been the trend since 1920, this year also the smaller animals made up the bulk of autopsies performed.

The manner of conducting the post mortems was that previously followed, namely: The students performed the autopsies under the instructor's supervision, while another student recorded the findings. Material for histological and bacteriological study was taken from every suitable case.

The total number of autopsies performed this year was 284. Of this number there were 14 horses, 21 cattle, 14 swine, 19 sheep, 129 dogs, 81 cats, 2 raccoons, 3 goats and 1 fox. As heretofore, the material was obtained from the college clinics and from practicing veterinarians from nearby towns.

In addition to the number recorded with the autopsies, we have received 50 specimens for diagnosis. Most of these were of neoplastic diseases and the so-called "infectious granulomata."

The following table gives the number and species of animals autopsied with the diseases that were directly or indirectly responsible for their death.

Table showing the number and species of animals autopsied during the year ending June 30, 1925, *with the diseases that were directly or indirectly responsible for their death.*

	Horses	Cattle	Swine	Sheep	Dogs	Cats
Abscess liver	1					
Anemia					1	1
Ankylostomiasis					2	
Arteriosclerosis		1			1	
Arthritis	1					
Ascariasis					1	2
Asphyxia					1	
Bronchitis				1		
Carcinoma					4	4
Colitis	1					2
Cystitis					1	
Distemper					52	
Encephalitis	1				1	
Enteritis		1	1		7	43
Epistaxis						1
Experimental			10	9		
Fatty liver					4	3
Foreign body tongue					1	
Fracture cranium	1					
Fracture forearm					1	
Fracture pelvis					1	
Gastric ulcer					3	2
Gastritis					2	
Gastroenteritis	1			1	5	1
Gonitis	1					1
Hemorrhage, internal	1				2	1
Hepatitis					1	
Hydronephrosis					1	
Indigestion, acute					1	

	Horses	Cattle	Swine	Sheep	Dogs	Cats
Intussusception					3	
Meningitis	1	2		1	1	1
Metritis		1		1	2	1
Myocardial degeneration					1	
Necrobacillosis				1		
Nephritis		1			2	3
Nutritional disease (stiffness)			2			
Parasitism					2	2
Pasteurellosis		5				
Pericarditis		1			1	
Peritonitis					2	2
Pneumonia	1	2		3	3	4
Poisoning (strychnine)					1	
Prolapse, intestine						1
Pulmonary oedema					1	
Pyemia					1	
Pyorrhoea					1	
Pyothorax					3	1
Rickets					1	
Rupture liver					1	1
Rupture uterus		1				
Senility	1				1	
Septicemia		3		2	1	1
Shock					1	
Stomatitis					4	
Suppuration						2
Taeniasis					1	1
Tetanus	2					
Torsion, ileum	1					
Traumatism					2	
Tuberculosis		3				
Uremia			1			
	14	21	14	19	129	81

In addition to the above, two racoons that died of peritonitis, three experimental goats and one fox that died of myocardial degeneration were examined, making a total of 284.

VITAL STATISTICS OF DISEASES OF THE GENITAL ORGANS OF COWS*

D. H. UDALL, E. R. CUSHING, AND M. G. FINCHER
Department of Medicine, Obstetrics, and Ambulatory Clinic

"If a problem is to be solved the nature of the problem and its difficulties should first be comprehended."—Mackenzie.[1] In the use of this principle we have learned that a study of many consecutive diseases of a system as they occur in the clinic reveals the importance of each, sometimes brings to light unsuspected facts, and often sifts out the speculative from the real. With this experience in mind compilations have been made upon 1000 affections of the generative organs of cows in an effort to learn more of the nature of the problem. Losses in this group due to partial or complete destruction of the reproductive functions, and to loss of life, far exceed those of any other animal disease in this part of the country.

The report covers the period from July, 1920, to December, 1924. It does not include all within the period, but it is representative of the group. The area covered is approximately fifteen miles in each direction, although most of our material is within a radius of ten miles. The information is about equally divided between grades and purebreds and is collected from many farms. The diagram in Fig. 1 is constructed from the 1,000 cases. Probably other observers and other localities would give some differences. Yet it is believed that it presents a useful means of estimating the nature and distribution of these affections. The observations are sufficiently accurate to enable deductions to be drawn, and they represent a survey that is far more complete than those upon which such estimates are usually made.

An introduction to diagnostic terminology shows that disease is named according to the symptoms, the pathological changes, or the etiology, and that even before Virchow founded cellular pathology or Pasteur discovered the microbian origin of disease men endeavored to visualize it in terms of etiology or of tissue change. For the most part we have followed the custom of naming the disease according to some outstanding symptom, such as abortion, retained placenta, sterility, etc. It is necessary, however, to consider another principle, which is also quoted from Mackenzie, "As disease is only made manifest to us by the symptoms it produces, to get a thorough understanding of disease, we must recognize the symptoms not merely by detecting their presence, but by understanding the mechanism of their production, and their bearing on the future health of the patient." To meet this conception, and to supply a pathological diagnosis we have let the circle itself, with

* Cornell Veterinarian, XV (1925), p. 121.

[1] Mackenzie, Sir James, *The Future of Medicine*, London, Henry Frowde and Hodder & Stoughton, 1919.

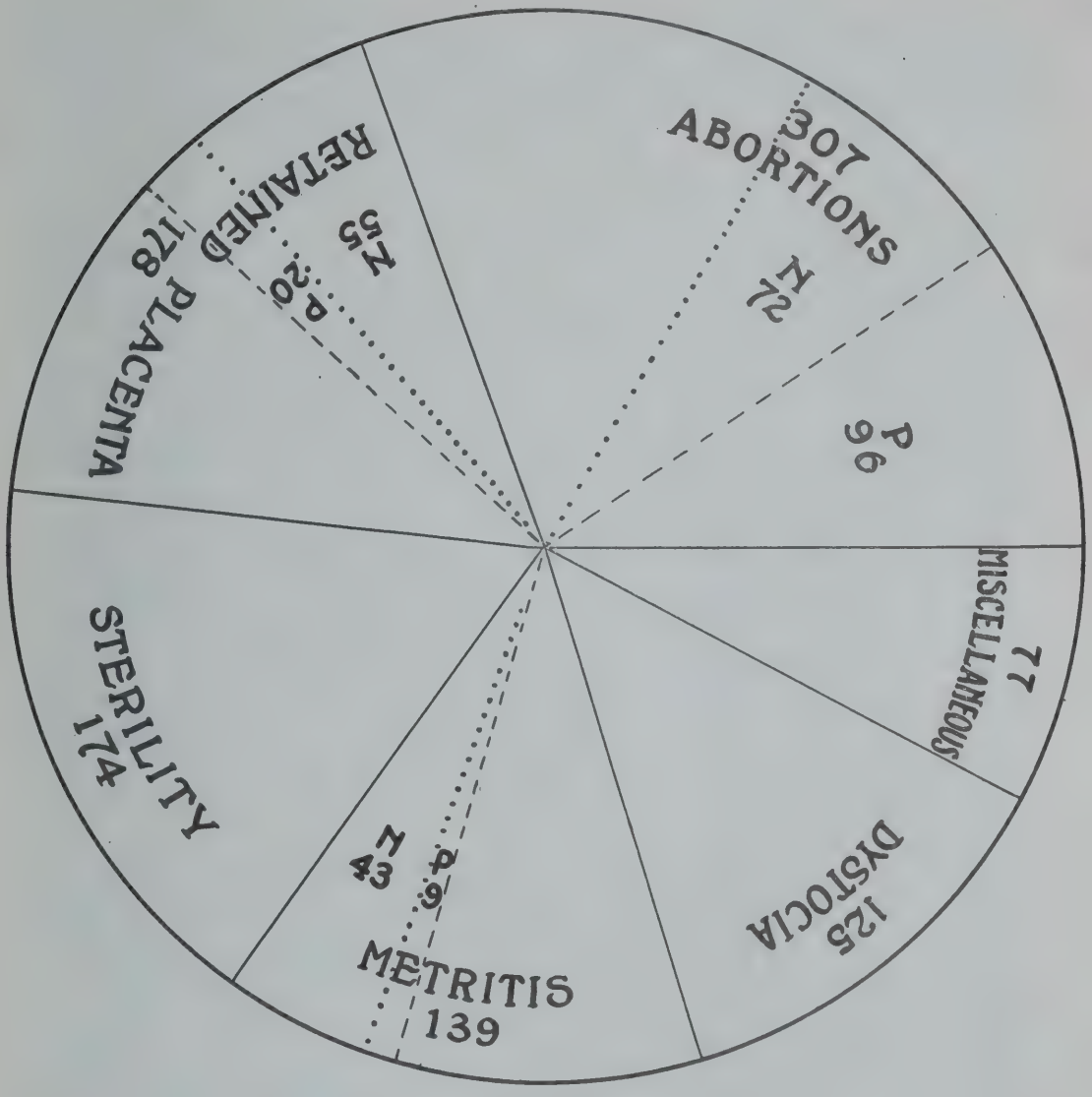


FIGURE 1

few breaks, stand as a legend for metritis. While it is recognized that the fetal membranes and the fetus in themselves constitute a physiological unit, the conception that they alone embrace the disease leaves the clinician with his sick patient in an awkward position. Further support of this premise is found in the fact that many of our patients have shown symptoms of disease even before conception. The presence of retained placenta in one-third of the total is a condition that is inconceivable except as a uterine disease. Thus we have, though using the language of symptoms, located the malady in the genital organs in terms of cellular pathology, and it is in these tissues or their appendages that one may logically seek for its microbian origin. From the uterus, disease often invades the placenta, less often the fetus. Differences in the structure of the uterus and placenta explain corresponding differences in the appearance of the lesions, which may be further modi-

fied in the latter by decomposition. A comparative study of the two reveals their interrelation, but the uterus itself remains as the essential organ to be considered in the prevention of genital disease and the repair of a damaged genital apparatus.

ABORTION

This group includes 307 in which a dead fetus or a premature sick calf was delivered. One hundred sixty-eight were examined by various methods for evidence of the presence of *Bacterium abortum,* Bang.[1] Of these, 96 or 57.2 per cent were positive, and 72 or 42.8 per cent were negative. Cows have been placed in the positive group on positive evidence obtained from either the blood, milk, feti, uterine exudate, or placentae. They have been placed in the negative group in the absence of such evidence on examination of the blood, milk, feti, placentae, etc. See Tables 1 and 2.

In the positive group are 89 blood examinations in which 12 or 13.5 per cent are negative in dilutions of 1 : 80 or more. Some of these are from negative herds, or occur under circumstances that indicate the permanent absence from the individual of a blood reaction. Forty-five milk examinations include 20 positives and 25 negatives. Thirty fetuses give 25 positives and 5 negatives. Fifty-three placentae give 49 positives and only 4 negatives. In some, the material examined consisted of uterine exudate rather than placenta.

When a bacterial disease is located in the uterus one would expect to find the cause in the placenta or the inflammatory exudate, and it is logical that this material should prove to be the most useful of all in revealing the presence of the Bang organism. Unfortunately the finding in this material of bacteria other than the Bang bacillus leads to an indefinite conclusion. If the Bang bacillus is found, it is the cause of the disease; when it is not found, the cause of the disease is unknown. The presence of other bacteria is sometimes explained on the theory that they might have entered the uterus through the cervical canal after the breaking of the uterine seal, and that they either established a primary infection, or contributed to one already present. That genital infection may occur in this manner is a well-known fact, but the frequency of such occurrence is not known. It is not rare to find a severe metritis in a cow that has an impervious uterine seal, and that is negative to the various tests for *Bact. abortum.* That genital infection other than *Bact. abortum* may be established in the sealed uterus is also a well-known fact. These other bacteria include the colon organism, the streptococcus, *B. pyogenes,* etc. Admitting that such bacteria may enter after the seal is broken and become the primary cause of metritis, or augment one that is already present, we still lack knowledge of their presence in the

[1] The laboratory work has been done chiefly by Dr. H. L. Gilman; in part by Doctors C. M. Carpenter, and W. A. Hagan.

uterus before the seal is broken. Such brief investigations as we have [1, 2] suggest that the pregnant uterus is the abode of various kinds of bacteria. A few carefully studied cases have shown that some of these bacteria may cause a metritis in the sealed uterus. With this principle established it is probable that the science of bacteriology will devise methods of establishing their relative importance, as well as knowledge of their time and manner of reaching the uterus.

The results of this compilation show that in more than half of the cases of acute metritis brought by us under laboratory examination, evidence of the presence of the Bang bacillus was wanting. Granting that in some it may have been missed, and that in others it may have vanished before the material reached our hands, there still remains an abundance clearly due to some other infection.

Bact. abortum present, blood negative. Occasionally *Bact. abortum* is found in the placenta or uterine exudate of a cow a few months before positive agglutinations are found in the individual or in the herd, and the blood of such an individual may remain permanently negative. This occurs in those that may or may not show symptoms of metritis. It suggests that the organism may be harbored by cows for months before its presence in the herd may be recognized by an examination of the blood.

Previous trouble in the abortion group. Incomplete as our reports are, they show a record of previous trouble in 20 per cent of the positives. In Table 3 are listed 25 cows that were diseased previous to abortion. Fifteen of the 25 were positives. This illustrates another principle; namely, *that disease progresses by a gradual development.*

Abortion or other trouble in more than one breeding year. Information on this point is incomplete because of so many recent additions; the observations are an index rather than an analysis. However, 86 animals, or 28 per cent of the 307, are included, and time will add several more.

The number of herds that contributed abortions are 142. Among these, 54 contain positives, and 36 negatives. Most of the negative farms comprise small herds from which only one sample of blood was taken. Whenever positive evidence is found on a farm it is listed as positive even though the dairy may have been negative for two or three years. A herd of any size in this area that remains entirely negative for more than two years is a striking exception.

Distribution according to the number of abortions in each herd indicates that enzootics constitute only a small per cent of the total.

---

[1] Carpenter, C. M. The bacteriology of the female reproductive organs of cattle and its relation to the diseases of calves, *Rep. N. Y. State Veterinary College*, 1920–21, p. 67.

[2] Hagan, W. A. The etiology and mode of infection in white scours in calves, *Rep. N. Y. State Veterinary College*, 1916–17, p. 156.

When one considers that this table is based on a period of four and one-half years it suggests that, in occurrence, the act of abortion regardless of the cause is an extremely common sporadic manifestation.

Number of abortions in herd....	1	2	3	4	5	6	7	8	9	10	12	16	20
Herds.....	79	26	11	5	2	1	1	2	1	1	1	1	1

This table does not include all of the abortions that occurred on each farm during the period, since many cases are ignored by the owner, especially in grade herds, unless other symptoms of metritis are also present. As a general indication, however, of the wide-spread nature of the condition, the figures are certainly helpful. These tabulations have brought out the variations in severity of genital disease on different farms, and on the same farm at different times. It is quite clear that relatively more disease exists on farms where the owner frequently buys and sells, on those that frequently change owners, and in the larger dairies. In other words, the rate and character of disease is changed when individuals from different sources are brought in contact. And the more intimate the contact between individuals on the same farm, the greater is the opportunity for disease.

AGE OF COWS AT TIME OF ABORTION

Years	Number	Per cent
2.....	66	22.22
3.....	51	17.17
4.....	55	18.51
5.....	30	10.10
6.....	39	13.13
7.....	19	6.39
8.....	12	4.04
9.....	6	2.02
Aged.....	19	6.39

Number of cows that aborted more than once: two times, 25; three times, 7; four times, 1; total, 33 = 11.11 per cent. Because so many have only recently come under observation, this percentage is considerably below the final.

AGE OF FETUS AT TIME OF ABORTION

Positives			Negatives		
Months	Number	Per cent	Months	Number	Per cent
1.....	.....	.....	1.....	.....	.....
2.....	.....	.....	2.....	1	1.75
3.....	2	2.29	3.....	1	1.75
4.....	1	1.15	4.....	4	7.02
5.....	7	8.04	5.....	5	8.77
6.....	18	20.69	6.....	6	10.52
7.....	20	22.99	7.....	10	17.54
8.....	34	39.08	8.....	13	22.81
9.....	5	5.75	9.....	17	29.82
	87			57	

Number of retained placentae in abortion group: 151, or 50 per cent. Among the positives are 51.6 per cent; among the negatives, 62.3 per cent.

Number of mummified fetuses: 10, or 3.25 per cent. Nine were negative, and one positive to *Bact. abortum.* The group contained 22 dystocias, and 19 pairs of twins. Among the latter were four positives, eight negatives, and ten at term.

In the herds in this series where abortion enzootics or storms have occurred in the older members of the herd as well as among the young, there has in each case been a history of careless breeding. This has taken the form of breeding the bull to the well and diseased, and to overwork of the bull. In both instances the initial symptoms were repeated breedings before conception was established, combined with minor changes in the vagina, the cervix, or the ovaries. To accurately assess the relative effect of an essential microbe and the numerous predisposing influences seems to us a difficult problem. But it is not difficult to recognize the presence of both, though much time may be required to escape from their effect.

Source of loss. The loss from abortion may be divided into two groups: immediate loss of milk and progeny, and a much greater loss of milk and progeny through impaired breeding function. The loss from deaths are 6, or 2 per cent; from condemnations 35, or 11.4 per cent; a course prolonged through more than one breeding year 86, or 28 per cent. When our information is all assembled the rate of all except immediate deaths will be still greater. Thus the chief loss is clearly the result of irreparable tissue changes in the uterus or its appendages. The prognosis is greatly influenced by the virulence of the infection, and the treatment before conception and after parturition. During the period

covered by these observations we have constantly had one or more herds under treatment where the virulence of the infection, regardless of its kind, seemed to determine a serious condition for the herd.

The solution of the problem consists in protection of the female by the adoption of all of the principles of hygiene that are logically sound even though their precise effect cannot be experimentally proven.

RETAINED PLACENTA

In addition to the 150 or more cases of retained membranes in the abortion group, there are 178 others in which the birth of a normal calf is reported. This total of over 300 shows the frequency of the condition, and the importance of knowledge of the pathology, the causes, and the treatment. It supports our conception that metritis is the fundamental lesion in most affections of this system, since retained placenta without metritis is a condition that we have never recognized.

What relation exists between retained placenta and infection with the Bang organism? In Tables 4 and 5 is a report of laboratory examinations on 75 of the 178 cases of this disease. Of these, 55 are negative, and 20 are positive. The placentae, however, are positive in only four of the nine in the positive group, which suggests that in some of these the Bang organism is absent from the uterus.

Other observations show that reproduction was destroyed in 8, or 4.5 per cent; and that death occurred in 6, or 3.3 per cent. In this series retained placenta alone seems to have been more fatal than abortion. This evidence supports the view that *Bact. abortum* infection is relatively mild. The view that retained placenta without abortion is a phase of Bang disease, or that it is evidence of previous infection with the Bang organism is not supported by the facts presented in Tables 4 and 5.

METRITIS

Acute metritis after normal delivery, with evidence of prompt expulsion of the fetal membranes, is found in 139. Eighteen died, and reproduction was destroyed in 8 others, a loss of 20 per cent—further evidence that diseases of the uterus are more serious in those infections that are less frequently associated with the Bang infection. In a total of 52 the negatives are 43, or 82.6 per cent. Individual herd records also indicate that acute or chronic affections not associated with *Bact. abortum* may be highly destructive to the genital organs. In this group are included two rather fatal types. The first is hydrops of the amnion, or allantois; the second, a septic metritis with a flaccid uterus that hangs forward over the pubic brim, contains large amounts of reddish pus, and is characterized clinically by milk-fever-like symptoms. Mild catarrhal endometritis is more frequent than the serious and destructive types. See Tables 6 and 7.

Sterility

Treatment of cows and heifers because of failure to breed included 174 animals, or 17.4 per cent. From veterinary literature one may easily receive the impression that sterility is for the most part the result of abortion, that it is allied to the *Bang abortion disease,* or that it is a phase of this disease. In the abortion group are listed all cases of sterility that have been known to follow abortion. It is probable that this list of 174 also includes a few among the older individuals that have previously aborted. The following age table of 153 animals in which 57.5 per cent are four years of age or younger indicates that on the whole the record in this respect is accurate.

Age Table 153 Animals

Age in years	2	3	4	5	6	7	8	9	Aged
Number of animals	38	27	23	16	17	5	12	2	13

An anatomical diagnosis in animals that fail to breed after repeated service is not always easy. Often several conditions may be found, any one of which is sufficient to account for the sterility. Our classification on 123 animals is as follows:

Estrum absent, buried corpus luteum	18.	
Cervicitis	21,	condemned 3.
Granular venereal disease	12.	
Cystic ovaries, nymphomaniac	10,	condemned 5.
Cystic ovaries, estrum absent	5.	
Cystic ovaries, estrum irregular	3,	condemned 1.
Cystic ovaries	2,	condemned 1.
Metritis, corpus luteum retained	6.	
Metritis, chronic	5,	condemned 4.
Metritis and cervicitis	4.	
Metritis, slight	4.	
Salpingitis	10,	condemned 8.
Ovaritis	8,	condemned 7.
No recognizable lesions	9,	condemned 1.
Estrum absent	4.	
Vaginitis	2.	

In a search for the causes of sterility one finds in the vagina, the cervix, and the uterus, evidence of infection to explain a considerable number. The nature of the infection, its origin, and manner of entry remain as in other groups, largely unexplained.

Distocia

In this group the age is rather striking. Fifty-five or 44 per cent are listed at two years or less. In 46 the symptoms indicated that distocia was preceded by a metritis. In the group of 125, thirteen died, a mortality of 10.4 per cent.

SUMMARY

A compilation of 1,000 consecutive cases of genital disease in cows gives 307 abortions, 178 retained placentae, 139 metritis (without abortion or retained placenta), 174 sterility, 125 dystocia, and 77 miscellaneous—the latter include torsion and prolapse of the uterus.

Laboratory examinations of 295 of the acute metritis group (abortion, retained placenta, and metritis alone) give the following results with respect to positive or negative evidence of association with *Bact. abortum:*

	+	—
Abortion	96	72
Retained placenta	20	55
Metritis alone	9	43
Total	125	170

TABLE 1.—ABORTION POSITIVES

Case	Blood [1]	Milk	Fetus	Placenta	Case	Blood [1]	Milk	Fetus	Placenta
40971	c80	—	+	+	56538	-20	+		
41114	c20	—		+	56840	c320			
41436 [2]	-20		+	—	57015	x320		+	+
41640	c500			+	57570	c640		+	
42647			—	+	57834	c640		+	+
44434			+		57849	-20			+
45511	c600				57855	x160	+		
45937				+	57857	x30	+	+	+
46928	c40	+	+		58072	c80			+
48045			—	+	58251			+	+
48168				+	58652	c640			—
48343		+			59318	c640	—		+
48352	c500				59744	c80			
48352	c500				59996	c240	—		+
48710	c500				60012	c640			
48734				+	60067	c240	—		+
49067	c500	+	—		60099	c640		+	
	c500				60432	c640			
	c640	+	+	+	60466	c640			+
49071 [3]	c80	—	+		60473	c40			+
49348	+	+	+		60762	x160	+		
49410 [4]	c600	+	+	—	60780	c640	+		
	c20 [5]	—		+	60780	c640			
49954	+				61104	c160	—		+
49963		+			61946	c640	+		
49966	+	—			62180	c640	—		+
	c640	—		+	62249	c320		+	+
50791					62295	c160			
52271	c640				62303	c640			
	c160				62308	-20	—		+
53004	c640			+	62800	c40	—	+	—
54158	c640				62865	c640	—		
54335	c20	—		+	62868	c640			
54420	c640	+	+		63074	c30	—		+
54575	x160				63229	c640	—		+
54577	x160				63761	c160	—		+
54759	x320				63835	c160	—		+
55238	c640	+	+		64075				+
55315	c40	—		+	64272	c160			
55552	c640		—	+	64373	c80	—		+
55884	c640				64496	c160	—		
55954	c320	+		+	64499	c320	+		+
55958 [6]					65797	c640			+
55964	c640		—	+	65173	c640	—		
56070	c640		+		65266	c640	+		+
56180	x320		+	+	65502	c320	+		+
56355	c640		+		65682	c640			
56356	c640		+	+	66293	c80		+	
56443	c640				66835	c640	—		+
56453	x320	+	+	+	67489				+
56454	c640	—		+					
	c160		+	+	96	89	45	30	53

[1] In this column c means complete, and x means partial agglutination.
[2] December 1921; October 1922,-20; November 1923,-20; October 1924, c20.
[3] 1924 [4] 1921. [5] 1924.
[6] Found in wall of uterus four months after abortion.

In Table 2 are listed the 72 cases of abortion in which laboratory examinations failed to reveal evidence of the presence of *Bact. abortum*.

TABLE 2.—ABORTION NEGATIVES

Case	Blood	Milk	Fetus	Placenta	Case	Blood	Milk	Fetus	Placenta
41436[1]	–20	.....	—	—	57746	c80	—	—	.....
42734[2]	–20	.....	—	—	58129	–20	.....	.....	.....
43006	c40	—	.....	.....	59225	–20	—	.....	—
42739[3]	c40	.....	.....	—	59230	–20	—	.....	.....
44896	–20	.....	.....	.....	59755	c20	—	.....	.....
45209	.....	.....	—	.....	59831	x40	—	.....	.....
45718	.....	.....	—	.....	60479	c80	.....	.....	.....
47696	.....	.....	.....	—	61009	–20	—	.....	—
48699	.....	.....	—	—	61106	–20	—	.....	.....
48921	—	—	.....	—	61148	–20	—	—	—
49135	—	.....	—	.....	62040	–20	—	.....	—
49687	–20	—	.....	—	62196	–20	—	.....	.....
49991	–20	—	—	—	62310	–20	—	—	.....
52335	c20	.....	.....	.....	62487	x40	.....	.....	.....
51443	–20	.....	.....	.....	62603	–20	.....	.....	.....
52970	–20	—	—	—	62672	c20	—	.....	.....
53162	–20	—	.....	—	62832	–20	—	.....	—
53378	.....	—	.....	—	62836	–20	—	.....	.....
53391	–20	—	.....	.....	62968	–20	.....	.....	.....
53633	.....	.....	.....	—	62970	–20	.....	.....	.....
54165	–20	—	.....	—	63102	x30	.....	.....	.....
54367	c50	—	.....	—	63506	–20	.....	.....	.....
54420	c20	.....	.....	—	63764	–20	.....	.....	.....
54429	.....	.....	—	.....	64976	–20	.....	.....	.....
55574	–20	—	.....	—	65227	–20	—	.....	—
55851	–20	—	.....	—	65664	–30	.....	.....	.....
55959	.....	.....	.....	—	65665	–20	.....	.....	.....
56036	c10	—	.....	—	66157	c20	—	.....	—
56059	–20	.....	—	.....	66529	c80	.....	—	.....
56068	.....	.....	—	.....	66591	c80	.....	.....	—
56146	–20	.....	—	.....	66691	c40	—	.....	.....
56172	–20	.....	—	.....	66697	c30	—	.....	.....
56354	c20	.....	—	.....	66836	c40	—	.....	—
56520	–20	—	—	—	66991	x30	.....	.....	.....
56542	–20	—	—	.....	67388	c40	—	—	.....
57094	—	.....	.....	.....					
57552	c40	.....	—	—	72	62	35	22	30

[1]February, 1923.

[2]Streptococcus isolated from two-months-old fetus.

[3]c40 in 24 hours;—20 in 30 days. 1924 aborted in Sept.; partial agglutination at 40 in December.

TABLE 3.—ABORTIONS, PREVIOUS TROUBLE

Case	Year	Age	Diagnosis
40971.....	1920	3	Calved.
	1921	4	Calved, metritis.
	1922	5	Calved, retained placenta.
	1923	6	Aborted.
41436.....	1919	2	Calved.
	1920	3	Calved, retained placenta.
	1921	4	Aborted at 8 mo.
	1923	6	Aborted at term, diffuse placenta.
	1924	7	Calved.
	1925	8	Calved, twin heifers, metritis.
42734.....	1917	8	Calved, milk fever, sterility.
	1918	9	Calved.
	1919	10	Calved.
	1920	11	Calved, metritis, sterility.
	1921	12	Aborted at 2 mo.
42739.....	1919	5	Calved twins, retained placenta.
	1920	6	Aborted twins, retained placenta.
	1921	7	Dystocia.
	1922	8	Aborted 6 mo.
	1924	10	Aborted twins.
44434.....	1917	5	Sterility.
	1920	8	Aborted, mummy.
45511.....	1920	5	Retained placenta.
	1921	6	Retained placenta, metritis, cervicitis.
	1922	7	Aborted 8 mo.
	1923	8	Salpingitis, killed.
46928.....	1920	6	Sterility.
	1921	7	Calved, chronic cervicitis, bred repeatedly.
	1924	10	Aborted.
48343.....	1921	5	Retained placenta.
	1923	7	Aborted.
49197.....	1920	3	Calved, metritis.
	1921	4	Calved, severe metritis.
	1922	5	Aborted, incurably sterile.
52970.....	1922	13	Calved.
	1922	13	Calved, metritis severe.
	1924	15	Aborted 5 mo., dropsy of amnion.
53004.....	1922	3	Granular vaginitis, sterility.
	1923	4	Aborted.
55315.....	1922	2	Calved, metritis, cervicitis.
	1923	3	Sterility.
	1924	4	Aborted.
55954.....	1922	2	Calved, metritis, cervicitis.
	1923	3	Aborted.
	1924	4	Aborted.

ABORTIONS, PREVIOUS TROUBLE

Case	Year	Age	Diagnosis
56180.....	1922	2	Sterility, bred repeatedly.
	1923	3	Aborted.
	1924	4	Aborted.
56355.....	1922	3	Sterility, bred repeatedly.
	1923	4	Aborted, twins at 8 mo.
	1924	5	Sterile, killed.
56451.....	1921	7	Calved, metritis, severe.
	1923	9	Aborted.
56453.....	1920	1	Sterility, bred repeatedly.
	1922	2	Sterility, bred repeatedly.
	1923	3	Aborted, incurably sterile.
56454.....	1921	4	Calved, bred repeatedly.
	1922	5	Sterility.
	1923	6	Aborted.
	1924	7	Aborted, incurable sterility.
56523.....	1923	3	Sterility.
	1924	4	Aborted.
	1925	.....	
58643.....	1921	2	Sterility, bred repeatedly.
	1923	4	Aborted.
59744.....	1921	4	Calved, bred repeatedly.
	1922	5	Aborted.
	1923	6	Calved, retained placenta.
59755.....	1923	6	Calved, retained placenta, metritis, cervicitis.
	1924	7	Aborted.
59996.....	1921	2	Sterility, bred repeatedly.
	1922	3	Calved.
	1923	4	Aborted.
60780.....	1923	2	Sterility, granular vaginitis.
	1924	3	Aborted.
64272.....	1923	3	Retained placenta.
	1924	4	Aborted.

TABLE 4.— RETAINED PLACENTA NEGATIVES

Case	Blood	Milk	Placenta	Case	Blood	Milk	Placenta
44466	.....	.....	—	59666	x40	—	—
46848	.....	.....	—	61360	—	—	.....
50253	—	.....	—	61941	—	—	.....
50801	—	.....	.....	62133	—	.....	.....
50920	—	—	—	62174	—	—	.....
51075	—	—	—	62254	c80	.....	.....
51339	—	—	—	62338	—	—	—
53383	—	.....	.....	62665	—	.....	.....
53958	–20	—	.....	62834	—	.....	.....
54426	c50	—	—	62976	—	—	—
54882	.....	—	—	63017	—	.....	.....
55271	–20	.....	.....	63026	—	—	.....
55313	—	.....	.....	63202	—	.....	.....
55334	–20	.....	.....	63763	—	.....	.....
55686	x40	.....	.....	63779	c40	—	—
56069	–20	.....	.....	63836	–20	—	—
56384	.....	.....	—	63827	c20	—	—
56689	–20	—	—	63896	–20	—	—
57090	–20	.....	—	64017	c40	—	.....
57091	–20	—	.....	64562	–20	.....	—
57098	c40	—	.....	65226	x80	—	—
57361	x40	—	—	65300	–20	—	—
57454	c40	.....	.....	65516	x60	.....	.....
57521	x20	.....	—	65844	—	—	—
57744	x80	—	—	66098	–20	—	—
57905	c40	—	.....	66377	–20	.....	.....
58135	–20	—	—	66599	c40	—	.....
58213	–20	.....	.....				
				55	51	30	27

TABLE 5.— RETAINED PLACENTA POSITIVES

Case	Blood	Milk	Placenta	Case	Blood	Milk	Placenta
47046 [1]	.....	.....	—	55783	c160	.....	—
47370	.....	.....	+	56897	c640	.....	.....
48467	+	.....	.....	58213	c640	—	—
48653	c640	.....	.....	60665	c640	.....	+
48996	+	.....	.....	62802	.....	+	—
48999	+	.....	.....	62131	c240	—	.....
49257	+	.....	.....	64566	–20	+	.....
49705	+	—	—	66096	c640	.....	+
50890	c640	.....	.....	66152	c160	.....	.....
54083	c200	—	.....				
55780	–20	—	+	20	17	7	9

[1] Calf died of scours, found positive.

TABLE 6.— METRITIS NEGATIVES

Case	Blood	Milk	Placenta	Case	Blood	Milk	Placenta
40381	–20	—	.....	61158	–20	—	—
44938	–20	.....	.....	61400	–20	.....	.....
49071	–20	—	—	62293	–20	.....	.....
48710	–20	—	—	62663	–20	.....	.....
49070	–20	.....	—	62884	c20	.....	.....
49196	–20	—	—	62901	c20	.....	.....
49197	–20	.....	.....	62958	–20	—	—
49198	–20	—	—	63955	–20	—	—
49467	–20	.....	.....	63129	.....	.....	—
50507	–20	.....	.....	63524	–20	.....	.....
50542	–20	.....	.....	63825	–20	—	.....
53005	–20	.....	.....	64073	–20	—	—
54526	c20	.....	.....	64298	–20	—	—
55314	–20	.....	.....	64299	c80	.....	.....
55846	–20	.....	.....	64814	–20	.....	.....
56684	c40	.....	.....	65010	–20	—	.....
56689	–20	—	—	65170	–20	.....	—
58883	–20	.....	.....	65820	–20	—	—
59742	.....	.....	—	66338	–20	—	.....
60058	–20	—	.....	67216	x40	.....	.....
60757	–20	—	.....	67215	x40	.....	.....
61012	–20	—	—				
				43	41	18	16

TABLE 7.— METRITIS POSITIVES

Case	Blood	Milk	Placenta
56827	c160	.....	+
56841	c320	.....	.....
57356	–20	—	+
59663	c320	.....	.....
60864	–20	—	+
60997	c640	.....	.....
62247	c160	.....	.....
63030	c30	+	+
63195	–20	.....	+
	9	3	5

These results indicate that in the control of genital disease clinicians are not justified in limiting their operations to those measures that are believed to control infection due to the Bang bacillus alone. The conception that other bacteria are largely secondary is a theory that receives slight support in the evidence obtained from this group of 1,000 cows.

PREVENTION OF DISEASES OF NEWBORN CALVES*

D. H. UDALL
Department of Medicine, Obstetrics, and Ambulatory Clinic

Modern studies of diseases of the newborn are based on the conception that bacteria are the main basic and determining cause of such afflictions, and classifications of such diseases according to the supposed etiology have been made. Two chief types are generally described: "White Scours," an intestinal condition for which the colon organism is held to be chiefly responsible; and "Pneumonia," for which the hemorrhagic septicemia group share the major blame. Additional study of the bacteriology of these diseases has involved so many varieties of bacteria that this phase of the problem is somewhat obscure. Time is required for a final decision.

At our conference last year Dr. Traum cited reports on the protective influence of colostrum when taken by the young. These show that protective antibodies against the bacterial associates of the dam are present in her colostral milk, and that such bodies are immediate protectants to the offspring that consume such milk. This seems to be nature's chief method of bacterial protection of the newborn. We have much to learn of the bacteriology of these diseases notwithstanding the important knowledge already acquired of the colon group and other types.

An important development of the knowledge that the colon organism and other bacteria were apparently the final influence in the destruction of the young was the artificial production of antibodies on a commercial scale. These are contained in white-scour serum and other similar products. They are supposed not only to protect against an invasion, but to overcome one that is already established. In this they seem to be fairly successful except where no affinity exists between the disease-producing body—the bacterium, and the antibody—the serum. In such cases immunity is established against bacteria that are not present. Some believe that any protection conferred by white-scour serum is due to the action of a foreign protein and not to a specific antibody. The impetus that gave rise to our feeding experiments was due in part to the apparent failure of white-scour serum, normal cow serum, and normal horse serum both as prophylactic and therapeutic agents. As a general rule it is not safe to substitute vaccination for other prophylactic methods. And this seems to be the tendency when it is adopted.

While the literature on the bacterial invasion of the young is abundant, on the subject of dietetics it is scanty. A few bulletins have been written on the economics of calf raising, but one will search long for specific information on any successful day-to-day

* Cornell Veterinarian, Vol. xiv, p. 226.

feeding program during the first few weeks of life. One might include a similar statement concerning our knowledge of colostrum and the physiology of digestion.

In the method of handling that I shall describe an effort has been made to begin with calves born at term in apparent health and establish a system of care and feeding that will maintain healthy vigorous growth for at least one month. With few exceptions the calves have come from herds where the mortality has been high. In each case the subject has been a Guernsey.

Any logical method of disease control is based on some theory or knowledge concerning its cause and nature. The term "white scours" as commonly used, means diarrhea during the first few days of the calf's life. In our experience this brief period represents the span of life of many calves born in apparent health. The first individual to die in a given season may belong to this type. A vigorous calf from a thoroughly segregated cow may be the only one to go. On autopsy the lesions are extremely variable. They may be slight. Pneumonia or gastro-enteritis are sometimes the dominant change. Others present diffuse subserous and submucous hemorrhages, while in some it is meningitis. The terms "white scours" and "pneumonia" are very well as far as they go, but when these are checked off many others remain. Just what per cent of these are septicemias due primarily to bacteria, and what per cent are intoxications due primarily to indigestion and presenting a terminal septicemia, I am uncertain. Our program has been formulated on the theory that nearly all are due to overeating within the first two or three days.

In older animals a knowledge of the cause of indigestion may reveal more with respect to the actual condition than one can learn from examination of the patient. When one draws a parallel with vigorous newborn animals sufficient theoretical reasons are found to explain many fatalities through indigestion. An animal with a ravenous appetite has access to an unlimited food supply. According to Smith and Little[1] the withholding of colostrum with antibodies leads to a fatal overwhelming with the colon organism. It seems plausible that an acute dilatation of the stomach from overloading may have a similar effect. Autopsies on cows that have died of indigestion often reveal tissue changes like those of septicemia; namely, extensive subserous hemorrhages, reddish fluid in the body cavities, enteritis, swollen glands, etc. There is evidence of an intoxication or septicemia that in this case we know to be terminal.

At parturition the modern dairy cow secretes milk in much greater quantities than did her progenitor. The digestive tract of her offspring has not undergone a compensatory development. Consequently it is more exposed to overloading. Indigestion is chiefly a functional disorder. The main characteristics are a food stasis associated with fermentation or putrefaction of the alimentary contents, deranged peristalsis and secretion, as well as various degrees

[1] The significance of colostrum to the newborn calf. Jour. Exp. Med., Aug. 1, 1922.

of intoxication and catarrh. It is a plausible theory at least that newborn calves, like other animals, may ingest more milk than they are capable of handling, and thus suspend the normal function of colostrum which performs the dual purpose of nutrition and immunization. This was suggested by the fact that in one herd calves removed from the dam at the end of thirty-six hours thrived, while those left for forty-eight hours nearly always suffered from scours.

The general appearance of milk at about the thirtieth hour after calving suggests that it is very rich in fat; only a small column at the bottom of a milk bottle resembles milk, the rest looks like cream. Recently the milk from cows before and after calving has been submitted for examination to Prof. P. A. Fish of the Department of Physiology. His report upon analyses of milk from two Guernsey cows is as follows:

Date of milking	Examined	Sp. Gr.	Coagulation	Colostrum corpuscles	Cream	Galactomicrons	Taste	Odor
12-26-23....	12-27-23	1035	—	—	4.60%	32.4	Normal....	Normal
1- 2-24....	1- 3-24	1036	—	—	4.05%	35.6	Normal.....	Normal
1- 3-24....	1- 4-24	1036	—	—	3.80%	32.4	Normal.....	Normal
1- 4-24....	1- 5-24	1035	—	—	.....	30.4	Normal.....	Normal
1- 7-24....	1- 8-24	1037	—	—	5.00%	32.2	Normal.....	Normal
1- 8-24....	1- 9-24	1036	—	—	5.80%	34.6	Normal.....	Normal
1- 9-24....	1-12-24	1037	ppt.	—	5.45%	23.0	Astringent..	Normal
1-10-24....	1-12-24	1039	—	—	5.15%	19.8	Astringent..	Normal
1-11-24....	1-12-24	1040	—	—	6.95%	24.6	Normal.....	Normal
1-11-24....	Calved..	....	.....	.....	.....	.....	.....	.....
1-13-24....	1-14-24	1035	—	—	6.60%	44.6	Normal....	Normal
1-14-24....	1-14-24	1037	—	—	4.10%	47.6	Normal.....	Normal
1-15-24....	1-16-24	1030	—	—	10.70%	50.4	Normal....	Normal
1-16-24....	1-17-24	1033	—	—	7.40%	27.4	Normal....	Normal
1-17-24....	1-18-24	1033	—	—	6.50%	34.8	Normal....	Normal
1-18-24....	1-18-24	1035	—	—	5.80%	38.6	Normal.....	Normal

The precipitate and astringent taste of January 9 and 10 milk might be due to a beginning souring. The milk was kept at outside temperature but was not examined until the third day after being taken from the cow.— P. A. FISH.

Date of milking	Examined	Sp. Gr.	Coagulation	Cream	Galactomicrons	Taste	Color
2-24-24 6:00 p. m.	Calved.						
2-25 8:30 a. m....	2-26	1053	Positive..	8.60%	48.0	Slimy....	Cream yellow
2-25 5:00 p. m....	2-26	1047	Slight....	9.60%	25.0	Slimy....	Cream yellow
2-26 8:30 a. m....	2-26	1043	Negative.	6.20%	56.6	Slimy....	Cream yellow
2-26 5:00 p. m....	2-27	1037	Negative.	5.65%	32.6	Slimy....	Cream yellow
2-27 8:30 a. m....	2-27	1036	Negative.	3.40%	30.8	Normal...	Normal
2-27 5:00 p. m....	2-28	1034	Negative.	6.50%	80.6	Normal..	Normal
2-28 8:30 a. m....	2-28	1035	Negative.	4.20%	39.6	Normal...	Normal
2-28 5:00 p. m....	2-29	1036	Negative.	5.10%	20.2	Normal..	Normal
2-29 8:30 p. m....	2-29	1036	Negative.	4.45%	22.4	Normal..	Normal
2-29 5:30 p. m....	3- 1	1035	Negative.	5.65%	76.8	Normal..	Normal
3- 1 8:30 a. m....	3- 1	1036	Negative.	4.45%	68.5	Peculiar..	Normal
3- 1 5:30 p. m....	3- 3	1036	Negative.	6.00%	98.2	Normal...	Normal
3- 2 8:30 a. m....	3- 3	1026	Negative.	4.50%	48.8	Normal...	Normal
3- 2 5:30 p. m....	3- 3	1036	Negative.	5.30%	75.4	Normal...	Normal
3- 3 8:30 a. m....	3- 3	1036	Negative.	4.60%	101.0	Normal..	Normal

My opinion is that the first two samples were quite decidedly in the nature of colostrum and that the subsequent samples were more like ordinary milk.— P. A. FISH.

To avoid what seemed to be overeating shortly after birth the following method was established:

In the beginning of the experiment each calf received soon after birth 40 to 60 cc. of normal cow serum subcutaneously. In a few this was administered in the vein, but after two or three collapsed and nearly died this method of administration was discontinued. It is not believed that the serum possesses any value. It was in use before this method of feeding was established and continued because of the report of Smith and Little[1] that it is an effective substitute for colostrum. Since calves normally receive colostrum from the dam no substitute is required.

METHOD OF FEEDING

1. For the first twelve hours the calf remains with the dam. During this period it obtains colostrum, but is not active enough to overeat.

2. At the end of twelve hours a muzzle is applied and all feed is withheld for twenty-four hours. Most calves show no evidence of extreme hunger, and when alone they sleep much of the time. Until the end of thirty days the muzzle is not removed except for feeding calves. Calves have individual characteristics. It is a common belief that a calf will not eat filthy bedding or other foreign material when in perfect health, and that the development of such a habit indicates the presence of disease. This is a fallacy. Some calves will begin to nibble straw when they are not more than three days old. When the muzzle is removed from others they may ravenously begin to chew the first thing at hand, such as another calf, the wall, or the bedding. These are the exceptions, but they are also the originators of trouble. A mass of hair, straw or shavings in the stomach is a foreign body that accomplishes no good purpose; it may cause the death of the individual, and serve as a starting point for disease among the others.

3. After thirty-six hours from birth the regular feeding routine is started. The allowance for the first feeding day is 5 to 6 per cent of the body weight of the calf of the dam's milk. Thus a 70-pound calf would receive about 3.5 pounds. This is divided into three feedings to each of which is added an equal amount of lime water. The mixture is heated to 100° F. and fed in a sterilized pail.

4. Immediately after the noon feed the calf is weighed and 6 or 7 per cent of this weight constitutes the ration of the dam's milk for the second feeding day. The amount of lime water added to each feeding is never more than one pint.

5. At the end of the first week the calf may receive 8 to 12 per cent of its body weight of milk daily. Feed in individual pails morning, noon, and night, each feed containing a pound of lime water. After feeding, rinse the pail in clean water, and scald it in

[1] Cow serum as a substitute for colostrum in newborn calves. Jour. Exp. Med., Oct. 1, 1922.

steam under pressure, or boil a small amount of water in it for two to five minutes. During the first week, before the noon feed, take the temperature. If it is 103° F., or more, give an enema and three ounces of liquid petrolatum, and withhold feed until the temperature is normal and the calf ravenously hungry. At birth give three ounces of liquid petrolatum to a 70-pound calf.

Housing conditions should protect calves against extreme cold, sudden changes in temperature, and cold drafts from open doors. They should be kept dry, warm, and clean. Each calf should be provided with an individual pen, but where muzzles are worn the dangers from mingling are very much reduced.

It is realized that all of the details outlined are not always essential or practical. But where disease is prevalent and the animals are valuable it is good economy to observe every detail.

It is believed that this method of care and feeding will prove satisfactory in calves that are strong and in good condition at the time of birth. When calves are prematurely born, or when they are very small, in poor condition, or weak, it is believed that the method should be modified to fit the individual. If it is unable to rise and suckle it may receive eight ounces of the dam's milk three to five times daily. In such individuals it is doubtful if the starvation period of twenty-four hours should be observed. At best, life hangs in a balance and no person can predict which way it will tilt. In one of the cases in this series the condition was worse at the end of the starvation period. One seems justified in the opinion that a continuous light diet is best adapted to weak calves.

A blood-tinged meconium seems to presage trouble. It indicates that the intestinal mucosa is diseased. This calf may receive two to four ounces of liquid petrolatum followed in a few hours by about ten grams of subnitrate of bismuth. Certain of these individuals seem to thrive better on a restricted milk diet supplemented by four ounces daily of olive oil. If the intestinal irritation becomes marked the diet may be restricted to olive oil until improvement takes place. Weakness may be combated with strychnine sulphate gr. 1/30 daily, omitted every third day. Other useful stimulants are aromatic spirits of ammonia ℥ iv in water ℥ viii, or camphorated oil 5 cc. in the pectoral muscles. The following tables illustrate the results in a few of the animals under this régime.

Bull No. 1 b. 9:00 a. m. 4–13–23, at 262 days				Heifer No. 2 b. 10:30 p. m. 4–18–23, at 267 days				Bull No. 3 b. 10:30 p. m. 4–19–23, at 284 days, wt. 70 lbs.			
Age in days	Wt. of calf	Fed per cent of body wt.	Pounds of milk daily	Age in days	Wt. of calf	Fed per cent of body wt.	Pounds of milk daily	Age in days	Wt. of calf	Fed per cent of body wt.	Pounds of milk daily
1	Unable to rise			1	Liq. pet. oz. iii			1	Liq. pet. oz. iii		
2	Down		2.0	2	69	.....	.....	2	Taken from dam		
4	Up, sucks			3	Taken from dam				at 18 hr., 5 p. m.		
	2 p. m. removed				at 36 hours			3	70	.....	1.0
5	45	11.0	5.0	4	.....	4.3	3.0	4	.....	4.0	3.0
6	.....	.....	6.0	5	.....	.....	3.0	5	.....	.....	3.0
7	.....	.....	6.0	6	.....	.....	3.0	6	70	.....	4.0
8	.....	.....	6.0	7	65	.....	4.0	7	71	7.0	5.0
9	.....	.....	6.0	8	65	7.7	5.0	8	74	8.4	6.0
10	.....	.....	6.0	9	67	9.2	6.0	9	74	9.5	7.0
11	53	.....	6.0	10	68	10.4	7.0	10	73	10.8	8.0
12	55	13.2	7.0	11	68	11.7	8.0	11	75	10.9	8.0
13	55	12.7	7.0	12	72	11.7	8.0	12	76	11.3	8.5
14	57	14.0	7.7	13	73	11.8	8.5	13	.....	11.8	9.0
15	58	14.5	8.5	14	.....	12.3	9.0	14	79	.....	10.0
16	58	13.7	8.3	15	75	.....	10.0	15	.....	12.6	10.0
17	61	14.3	8.3	16	.....	.....	10.0	16	81	.....	10.0
18	63	14.2	8.7	17	76	.....	10.0	17	.....	.....	10.0
19	.....	14.3	9.0	18	.....	.....	10.0	18	.....	.....	10.0
20	65	.....	10.0	19	.....	.....	10.0	19	.....	.....	10.0
21	.....	.....	10.0	20	.....	.....	10.0	20	83	.....	10.0
22	66	15.4	10.0	21	78	.....	10.0	21	85	12.0	10.0
23	.....	.....	10.0	22	81	12.8	10.0	22	88	13.0	11.0
24	.....	.....	10.0	23	82	13.5	11.0	23	90	12.5	11.0
25	.....	.....	10.0	24	84	13.4	11.0	24	90	12.2	11.0
26	70	.....	10.0	25	84	13.0	11.0	25	.....	.....	11.0
27	71	14.3	10.0	26	.....	.....	11.0	26	94	.....	11.0
28	73	15.3	11.0	27	86	.....	11.0	27	.....	.....	12.0
29	76	15.0	11.0	28	.....	12.0	12.0	28	97	.....	12.0
32	80	.....	.....	29	90	.....	12.0	29	.....	.....	12.0
....	.....	.....	.....	30	.....	.....	12.0	30	.....	.....	12.0
....	.....	.....	.....	34	99	.....	.....	33	106	.....	13.0
....	.....	.....	.....	.....	.....	.....	.....	39	116	.....	.....

Bull No. 4 b. 11:00 a. m. 4–24–23, at 282 days				Heifer No. 5 b. 5–3–23, at 283 days				Bull No. 6 b. 6:00 p. m. 9–15–23, at 279 days			
Age in days	Wt. of calf	Fed per cent of body wt.	Pounds of milk daily	Age in days	Wt. of calf	Fed per cent of body wt.	Pounds of milk daily	Age in days	Wt. of calf	Fed per cent of body wt.	Pounds of milk daily
1	82	.....	.....	1	Taken from dam at 12 hours			1	Taken from dam at 12 hrs., 12 p. m.		
2	85	Taken from dam at 21 hr.,		2	Refused milk			2	.....	.....	1.0
	8:00	a. m.		3	59	5.3	3.0	3	72	5.0	3.6
3	84	4.7	4.0	4	57	7.0	4.0	4	73	7.0	5.0
4	84	7.1	6.0	5	60	12.7	7.0	5	75	8.9	6.5
5	83	8.4	7.0	6	61	13.0	7.8	6	77	11.0	8.2
6	87	8.4	7.0	7	.....	13.0	8.0	7	80	12.0	9.2
7	88	8.5	7.5	8	.....	.....	8.2	8	83	12.0	9.6
8	.....	.....	.....	9	63	13.0	8.2	9	85	13.0	10.8
9	90	10.2	9.0	10	.....	13.0	8.2	10	.....	14.0	12.0
10	.....	.....	9.0	11	.....	.....	8.2	11	89	.....	12.0
11	90	10.2	9.0	12	66	.....	8.0	12	91	13.5	12.0
12	.....	.....	9.0	13	.....	.....	8.7	13	.....	13.0	11.8
13	.....	.....	9.0	14	.....	.....	8.7	14	94	.....	12.0
14	.....	.....	9.0	15	.....	.....	9.0	15	.....	.....	12.0
15	92	.....	9.0	16	.....	.....	9.2	16	.....	.....	12.5
16	94	9.7	9.0	17	.....	.....	11.2	17	.....	.....	12.5
17	96	10.4	10.0	18	.....	.....	12.0	18	101	15.0	15.0
18	97	10.6	10.0	19	.....	.....	12.0	19	105	15.0	15.1
19	97	10.4	10.0	20	80	.....	13.0	20	109	15.0	15.0
20	.....	.....	10.0	21	.....	.....	13.0	21	110	15.0	16.3
21	101	.....	10.0	22	.....	.....	13.5	22	.....	15.0	16.5
22	.....	10.8	11.0	23	.....	.....	13.5	23	.....	.....	16.5
23	103	.....	11.0	24	.....	.....	13.5	24	117	.....	16.6
24	.....	.....	11.0	25	.....	.....	15.5	25	120	15.0	17.5
25	.....	.....	11.0	26	.....	.....	15.7	26	122	15.0	18.0
26	.....	.....	11.0	27	.....	.....	16.5	27	126	14.7	18.0
28	110	.....	13.0	28	.....	.....	16.0	28	129	15.0	18.9
33	121	.....	13.0	29	.....	.....	16.0	29	133	15.0	19.5
....	.....	.....	.....	30	95	.....	16.0	30	134	15.0	19.8
....	.....	.....	.....	.....	.....	.....	.....	31	137	15.0	20.1
....	.....	.....	.....	.....	.....	.....	.....	32	141	15.0	20.5

Heifer No. 7 b. 6 :30 p. m. 11–3–23, at 286 days			
Age in days	Weight of calf	Fed per cent of body weight	Pounds of milk daily
1	64 Liq. pet. oz. iv. Taken from dam at 12 hr., 6 p. m.		
2	60	.....	1.0
3	57	4.2	2.5
4	59	7.0	4.0
5	59	7.6	4.5
6	61	8.5	5.0
7	62	9.0	5.5
8	63	10.0	6.2
9	.....	12.0	7.5
10	66	.....	7.5
11	68	13.0	8.5
12	70	14.0	9.5
13	72	15.0	10.0
14	73	15.0	10.8
15	76	15.0	11.0
16	.....	15.0	11.5
17	80	.....	11.5
18	82	15.0	12.0
19	84	15.0	12.5
20	86	14.9	12.5
21	87	15.0	13.0
22	87	15.0	13.0
23	.....	.....	13.0
24	91	.....	13.0
25	93	15.0	13.6
26	95	15.0	14.0
27	.....	14.7	14.0
28	100	.....	14.2
29	101	14.2	14.2
30	.....	.....	14.2
31	105	.....	14.5
33	110	.....	15.0

Bull No. 8 b. 5 :00 p. m. 10–19–22, at 263 days weight, 47 pounds	
2	Taken from dam at 6 a. m., 13 hr.
3	Starved. Seems normal
4	9 :00 a. m., eyes sunken. T. 97 F. Unable to eat, died in p. m. Autopsy, negative

Heifer No. 9 b. 9 :00 a. m. 11–7–23, at 280 days			
Age in days	Weight of calf	Fed per cent of body weight	Pounds of milk daily
1	80 Liq. pt. oz. iv. Taken from dam at 11 hr., 8 p. m.		
2	78	.....	.....
3	76	5.0	4.0
4	77	7.0	5.3
5	.....	9.0	7.0
6	80	.....	7.0
7	82	11.0	8.8
8	84	12.0	10.0
9	88	14.0	11.7
10	91	14.0	12.3
11	94	15.0	13.6
12	.....	.....	14.0
13	98	.....	14.0
14	100	15.0	14.7
15	103	15.0	15.0
16	106	.....	16.0
17	107	15	16.0
18	109	15	16.1
19	.....	.....	16.1
20	113	.....	16.1
21	115	15	17.0
22	118	15	17.2
23	.....	14.6	17.3
24	121	.....	17.7
25	125	15.0	18.0
26	.....	14.4	18.0
27	129	.....	18.0
28	.....	14.0	18.0
29	133	.....	18.0
30	134	13.5	18.0
31	137	13.4	18.0
..	.....	.....	.....

Heifer No. 10 b. 9:00 a. m. 1–11–24, at 280 days				Heifer No. 11 b. 6:00 p. m. 2–24–24, at 274 days. Weak			
Age in days	Weight of calf	Fed per cent of body weight	Pounds of milk daily	Age in days	Weight of calf	Fed per cent of body weight	Pounds of milk daily
1		Taken from dam at 12 hr., 9 p. m.		1	.....	.....	.3
2	65	Weak, gaunt, bloody meconium		2	.....	.....	1.6
	.....	.....	1.0			3:00 p. m., able to stand	
3	.....	4.6	3.0	3	.....	.....	3.0
4	63	.....	1.5	4	53	.....	3.0
5	.....	.....	1.5			Taken from dam at 74 hrs.	
6	61	5.0	3.0	5	55	6.6	3.5
7	61	5.0	3.0	6	57	7.4	4.1
8	62	4.8	3.0	7	60	8.2	4.7
9	60	4.2	2.5	8	.....	9.1	5.5
10	.....	.....	3.0	9	58	Scours	3.0
11	62	5.6	3.5	10	56	4.1	2.5
12	63	7.1	4.5	11	57	5.3	3.0
13	63	7.1	4.5	12	.....	6.1	3.5
14	63	2.4	1.5	13	.....	.....	3.8
15	60	4.0	2.4	14	.....	.....	4.2
16	60	7.6	4.6	15	.....	.....	4.3
17	.....	.....	3.7	16	56	.....	4.4
18	61	5.0	3.0	17	57	8.2	4.6
19	60	6.6	4.0	18	.....	8.4	4.8
20	60	5.5	3.3	19	.....	.....	5.0
21	61	7.1	4.3	20	60	.....	5.4
22	61	7.8	4.8	21	.....	9.3	5.6
23	63	8.2	5.0	22	.....	.....	5.9
24	.....	9.5	6.0	23	.....	.....	6.0
25	67	.....	6.2	24	.....	.....	6.2
26	70	10.4	7.0	25	64	.....	6.7
27	.....	9.7	6.8	26	66	12.8	8.2
28	.....	.....	4.1	27	69	14.0	9.3
29	71	7.7	5.5	28	.....	.....	9.7
30	.....	10.4	7.4	29	74	.....	10.0
31	.....	.....	8.0	30	75	15.1	11.2
32	.....	.....	6.0	31	77	14.9	11.2
33	.....	.....	6.0	32	80	15.0	11.7
34	71	11.2	8.0				
35	72	14.0	10.0				
36	.....	13.9	10.0				
37	.....	.....	4.0				
38	.....	.....	1.0				
39	Dead	.....	.....				

Remarks: Heif. No. 10, b. 1/11/24 at 282 days, wt. 65 lbs. The dam was a 4-yr-old cow that aborted in 1922, calved at 282 days in 1923, and broke the record for her class in pounds of milk produced while carrying this calf. The placenta came away after forty-eight hours. Some have suggested that the fetus is adversely influenced when the dam produces heavily, but the calves in this series do not support that view.

While this calf weighed 65 lbs. at birth, and was born at term, it was weak and gaunt, and the meconium was streaked with blood. It had suckled the dam freely. On the third day the usual amount of equal parts of lime water and milk were fed and nothing abnormal was noted.

4th day. Severe fetid diarrhea observed after morning feed.

5th day. Calf emaciated and weak, feces pasty and blood-tinged. Strych. sulph. gr. 1/15 at 4:00 and 7:30 p. m., *per os.* Fed at 7:30 a. m., and 2:30 p. m.

6th day. 7:30 a. m., refused milk. 11:30 a. m. adm. 1 lb. ea. milk and lime-water with syringe. 12:00 m., adm. spts. ammon. arom. ℥ iv in water ℥ viii. 1:45 p. m., adm. olive oil ℥ iv. Evacuations firm, white. Stronger and brighter than this a. m. 3:30 p. m., fed 1 lb. ea. milk and lime water—syringe. Adm. olive oil ℥ iv; strych. sulph. gr. 1/15. 10:00 p. m., fed 1 lb. ea. milk and lime-water—drank.

7th day. Fed 1 lb. ea. milk and lime-water—drank. 10:00 a. m., adm. strych. sulph. gr. 1/15. 2:00 p. m., fed 1 lb. ea. milk and lime-water—drank one-half. Adm. olive oil ℥ iv. Brighter. 8:00 p. m., fed 1 lb. ea. milk and lime-water—drank.

8th day. 7:00 a. m., fed 1 lb. ea. milk and lime-water—drank. 10:45 a. m., hungry, active, calling for food. Fed 8 oz. ea. milk and lime-water—drank. Adm. olive oil ℥ iv. 2:00 p. m., fed 1 lb. ea. milk and lime-water—drank nearly all. Adm. strych. sulph. gr. 1/15. Active. 4:30 p. m., adm. olive oil ℥ iv. Playing. 8:00 p. m., fed 1 lb. ea. milk and lime-water—drank.

9th day. 7:15 a. m., fed 1 lb. ea. milk and lime-water—drank half, fed rest with syringe. 3:00 p. m., same as this morning. Adm. olive oil ℥ iv; strych. sulph. gr. 1/15. Less active than yesterday. Saline enema returns much mucus. T. 101.4. 7:30 p. m., fed 8 oz. ea. milk and lime-water, and 4 oz. olive oil with syringe.

10th day. 8:00 a. m., fed 1 lb. ea. milk and lime-water—drank. 9:30 a. m., adm. strych. sulph. gr. 1/15. 2:40 p. m., fed 1 lb. ea. milk and lime-water—drank; 8:30 p. m., repeated.

13th day. Gradually improving. Wesson's cooking oil ℥ v was substituted for olive oil with the object of increasing nutrition. At 8:45 p. m. the appetite was poor.

14th day. Fetid severe diarrhea this a. m. 7:15 fed 1.5 lb. milk. 8:00 a. m., strych. sulph. gr. 1/15. T. 102.6. 9:30 a. m., saline enema. Adm. spts. ammon. arom. ℥ iv. Weak. 11:45 a. m., spts. ammon. arom. ℥ iv. 1:30 p. m., fed 8 oz. ea. pasteurized milk and lime-water. 2:15 p. m., colicy pains. Palpation of abdomen reveals an abundance of liquid in the stomachs. Grating teeth. 3:30 p. m., strych, sulph. gr. 1/15. Feces liquid. 8:00 p. m., fed 8 oz. pasteurized milk without lime-water—drank. Adm. bismuth subnitrate gm. x. The lime-water was omitted because it was thought to contribute to the distension of the stomach with liquid. This change with the addition of bismuth was made because of failure to produce consistent gain. Improvement in the strength and attitude was noted within twenty-four hours. T. 102.4.

15th day. 7:00 a. m., fed 8 oz. past. milk—drank. 10:30 a. m., adm. strych. sulph. gr. 1/15; bismuth subnitrate gm. x. Feces thin, black, condition poor, weak. 1:30 p. m., fed 10 oz. past. milk—drank greedily. Abdomen smaller. T. 103 F. 5:00 p. m., fed 10 oz. past. milk. Adm. bismuth subnitrate gm. x. 8:00 p. m., fed 12 oz. past. milk, adm. strych. sulph. gr. 1/15.

16th day. Hungry. Improved. Adm. bismuth subnitrate gm. x.
19th day. Hardly able to rise. T. 103.4.
20th day. Hungry and active.
36th day. About noon the calf was found down, unable to rise, the abdomen distended with liquid in the stomachs, no diarrhea, but still hungry. Treatment failed. For the previous two weeks the calf had been improving. Pasteurized milk in four to five feedings daily was continued until the 29th day, when a change to raw milk at 100 F. was made. The final and fatal relapse was clearly the result of a violation of our own rules with respect to feeding. On the 34th day the milk was increased two pounds, and on the 35th an equal amount.

From the time that the lime-water was omitted, bismuth subnitrate given in large doses, and pasteurized milk fed five times daily, improvement was constant. While this calf never gave promise of possessing economic value—she was always emaciated and somewhat weak behind—there is hardly a doubt of her survival had the increase in milk been more gradual. *Autopsy:* Negative with the exception of degeneration of the liver.

Heifer No. 11, b. 2/24/24, at 274 days, weak and unable to rise and stand until about 18 hours old. The dam was born in October 1913. Her available history is as follows: May 1915, treated for metritis following abortion; Sept. 1915, found pregnant; 1920 calved; 1921 calved, calf weak and lived three days; 2/27/23, calved, calf normal and healthy but died when about a month old.

1st day. Adm. 2 oz. olive oil and strych. sulph. gr. 1/30.
2nd day. 8:00 a. m., adm. 2 oz. olive oil and strych. sulph. gr. 1/30. Hungry, suckles cow when helped to stand. 3:00 p.m., adm. 2 oz. olive oil. 8 p. m., adm. 2 oz. olive oil and strych. sulph. gr. 1/30. Muzzled.
9th day. Scouring badly, white, pasty and semifluid feces, active, stomachs distended with fluid. Treatment consisted in reduction of milk as shown in table, and giving three to eight ounces of olive oil daily for three days. This animal belongs to that group of individuals that seem to require constant nourishment from birth. It is interesting to note that in the last twelve days of the record it made a gain of 20 lbs.

Bull No. 8. Without considering that this calf was underweight and premature, he was placed on the same régime as his predecessors in this series, with the exception of No. 1. His survival under any circumstances was questionable. On autopsy only a small clot of milk was found in the stomach, and it is possible that he obtained very little from the dam. No. 1 was physically less promising on the second day, but he received milk in small amounts frequently.

CONCLUSIONS

1. The object of this work has been to learn the best method of handling newborn calves in herds where losses occur. It has been conducted on the theory that improper feeding and not infection is the basic cause of the trouble. It is recognized that a

sick calf, under any circumstances, may originate destructive infections when it mingles with others.

2. Calves born at full term, strong, vigorous, and in apparent health often sicken and die with or without symptons of "scours" after a period of about three days. It is believed that a ravenous appetite and an unlimited milk supply from the dam are responsible for an overloading of the stomach with a subsequent indigestion and a terminal fatal intoxication. It is believed that fatalities may be reduced by following the methods described on page 70 under "Method of Feeding."

3. Calves that at birth are weak, or poor, or undersized, or premature—265 days—require special care and feeding. Instead of obtaining too much of the dam's milk there is danger that they receive too little unless it is milked and fed to them with a syringe. They should be fed not less than three times in each twenty-four hours. When regular feeding is established, the daily per cent of increase should be less than in strong individuals.

STUDIES OF GENITAL DISEASE IN A HERD OF DAIRY CATTLE*

M. G. FINCHER

Department of Medicine, Obstetrics, and Ambulatory Clinic

GENERAL DISCUSSION

Since the discovery by Bang of the *Bacterium abortum* in 1896 it has been regarded as one of the chief obstacles to reproduction among cattle. Some speculation has arisen over what might occur in a herd in the absence of such infection. In 1920 Herd 1, a Holstein dairy of thirty breeding cows located near the college, was the object of visits for the treatment of metritis. Numerous laboratory examinations of blood, placentae, and aborted fetuses invariably failed to show any evidence of the Bang organism. Finally, late in 1924, evidence of such infection became abundant. This paper is the record of the herd from 1920 until March, 1925. It was practically free from disease due to *Bact. abortum* from October 1922 to October 1924, and it is probable that it was free from the infection for a considerable period before the negative blood test of October 26, 1922. Finally, the Bang organism gained entrance, and the final record is one of that infection superimposed upon the previous condition.

Since 1922 limited observations have been made upon thirty-four heifers which have not yet calved or were sold before parturition. Two bulls, "A" and "B", were the only animals not raised from foundation stock, and were bought when 7 months old.

TABLE 1 — HERD AGGLUTINATION TESTS

Year	Month	Cows	Heifers	Remarks
1922	Oct.	32	9	One was partial at 1:20
1923	Jan.	29	.....	Six were partial at 1:20
1923	May	21	.....	One slight at 1:80 Seven complete at 1:40 Three slight at 1:40 One complete at 1:20
1923	Nov.	24	7	All negative in all dilutions.
1924	Mar.	3	.....	One slight at 1:120 One slight at 1:30
1924	May	27	.....	All negative in all dilutions.
1924	Sept.	28	12	Twenty entirely negative. Fourteen complete at 1:20 Three complete at 1:40 Two complete at 1:80 One complete at 1:120

* The laboratory examinations were made by Drs. C. M. Carpenter, H. L. Gilman, and W. A. Hagan.

Year	Month	Cows	Heifers	Remarks
1924	Dec.	16	8	One entirely negative. Four complete at.....1 :20 Five partial at.....1 :40 One complete at.....1 :40 Two partial at.....1 :80 Eleven complete at 1 :80 or higher
1925	Jan.	13	20	Seven entirely negative. Two complete at.....1 :20 Eight slight at.....1 :40 Three complete at.....1 :40 Two slight at.....1 :80 Eleven complete at 1 :80 or higher

TABLE 2—INDIVIDUAL AGGLUTINATION TESTS

Cow's number	10/26 '22	1/17 '23	5/30 '23	11/27 '23	3/11 '24	5/27 '24	9/19 '24	12/12 '24	1/7 '25	1/19 '25
10 (also Case No.)	—	—	....	—	....	—	c40	....	x80	....
11 "	—	—	—	—	....	—	c20	x40	c80	c80
12 "	—	—	x40	—	....	—	—	....	....	....
13 "	—	—	c40	—	....	—	c20	x80	....	....
14 "	—	—	c40	....	....	—	c20	c20	....	....
15 "	—	—	x80	—	....	—	c20	x80	....	....
16 "	—	—	c40	—	....	—	—	....	c80	....
17 "	—	x20	—	....	....	—	—	....	....	....
18 "	—	x20	x40	—	x30	Killed	....	....	....	....
19 "	x20	Died	....	....	....	....	....	....	....	....
20 "	—	—	Died	....	....	....	....	....	....	....
21 "	—	Sold	....	....	....	....	....	....	....	....
22 "	—	—	....	—	....	—	—	x160	....	....
23 "	—	—	—	—	....	—	c20	x320	....	....
24 "	—	—	—	—	....	—	—	....	x40	....
25 "	—	....	Sold	....	....	....	....	....	....	....
26 "	—	—	c20	—	....	—	—	c40	....	....
27 "	—	x20	—	—	....	—	—	c80	x320	....
28 "	....	....	....	....	....	....	—	....	—	....
29 "	....	—	....	—	....	—	—	....	c80	c80
30 "	—	—	—	—	....	—	c20	....	c320	....
31 "	—	x20	c40	—	x120	Died	....	....	....	....
32 "	—	—	....	—	....	—	—	c20	....	....
33 "	—	—	—	—	....	—	c20	x640	....	....
34 "	—	x20	c40	—	....	—	c120	c640	....	....
35 "	—	—	x40	—	....	—	....	c80	....	....
36 "	—	—	—	—	....	—	c80	x40	x80	—
37 "	....	....	....	....	....	....	c20	x160	....	....
38 "	....	....	....	....	....	....	—	x160	x240	....
39 "	—	—	c40	—	....	—	—	....	c640	....
40 "	—	x20	c40	Sold	....	....	....	....	....	....
41 "	—	—	—	—	....	—	c20	c160	....	....
42 "	—	—	—	—	....	....	....	....	....	....
43 "	—	—	—	—	....	....	—	....	....	....
44 "	—	Sold	....	....	....	....	....	....	....	....
45 "	—	Sold	....	....	....	....	....	....	....	....
46 "	....	—	Sold	....	....	....	....	....	....	....
47 "	....	....	....	....	....	....	—	....	....	....
48 "	....	....	....	....	....	—	c20	....	x640	....
49 "	....	....	....	....	....	....	—	....	c80	....
HEIFERS										
50 "	—	....	....	—	....	....	....	....	....	....
51 "	—	....	....	—	....	....	....	....	....	....
52 "	—	....	....	—	....	....	....	....	....	....
53 "	—	....	....	—	....	....	....	....	....	....
55 "	—	....	....	—	....	....	....	....	....	....
55 "	—	....	....	....	....	....	....	....	....	....
56 "	—	....	....	—	....	....	....	....	....	....
57 "	—	....	....	....	....	....	....	....	....	....
58 "	—	....	....	....	....	....	....	....	....	....
59 "	....	....	....	—	....	....	—	....	x160	....

Cow's number	10/26 '22	1/17 '23	5/30 '23	11/27 '23	3/11 '24	5/27 '24	9/19 '24	12/12 '24	1/7 '25	1/19 '25
60 "	....	....	....	....	....	....	....	—	—	....	....
61 "	....	....	....	....	....	....	....	c40	....	c20	....
62 "	....	....	....	....	....	....	....	—	x40	....	....
63 "	....	....	....	....	....	....	....	c20	x40	....	....
64 "	....	....	....	....	....	....	....	c20	c80	....	....
65 "	....	....	....	....	....	....	....	c20	c20	....	....
66 "	....	....	....	....	....	....	....	c20	c20	....	....
67 "	....	....	....	....	....	....	....	—	....	—	....
68 "	....	....	....	....	....	....	....	c40	....	c40	....
69 "	....	....	....	....	....	....	....	c80	c80	....	....
70 "	....	....	....	....	....	....	....	—	x40	....	....
71 "	....	....	....	....	....	....	....	....	....	x40	....
72 "	....	....	....	....	....	....	....	....	....	x40	....
73 "	....	....	....	....	....	....	....	....	....	x40	....
74 "	....	....	....	....	....	....	....	....	....	c640	....
75 "	....	....	....	....	....	....	....	....	....	x40	....
76 "	....	....	....	....	....	....	....	....	....	—	....
77 "	....	....	....	....	....	....	....	....	....	—	....
78 "	....	....	....	....	....	....	....	....	....	—	....
79 "	....	....	....	....	....	....	....	....	....	x40	....
80 "	....	....	....	....	....	....	....	....	....	x40	....
81 "	....	....	....	....	....	....	....	....	....	c40	....
82 "	....	....	....	....	....	....	....	....	....	—	....
83 "	....	....	....	....	....	....	....	....	....	c40	....
84 "	....	....	....	....	....	....	....	....	....	x40	....
85 "	....	....	....	....	....	....	....	....	....	—	....
86 "	....	....	....	....	....	....	....	....	....	c20	....

c Means complete agglutination at the given dilution.
x Means partial or slight agglutination.

Bull "A" was negative in October 1922, and was not used after March 1923. Bull "B" was negative on six or more tests given at the time of the herd tests. These tests include forty cows and thirty-seven heifers. Three of the latter became cows and are so listed in Table 2.

HERD BULLS

The following brief history of the two bulls used in this herd shows that the data regarding them are too incomplete to prove that they played an important part in causing the disease. Both were used upon cows from Herd II, another Holstein dairy which was located two miles distant. This herd became definitely infected with *Bact. abortum* a year earlier than Herd I. Whether the infection was carried from the farm of Herd II by the bulls cannot be determined, but that was the only known source of contact with outside animals.

Bull "A," born Feb. 27, 1916, was purchased at 7 months of age. He was first used here for service when about 1 year old. During the next 5 years he was bred to cows from Herds I and II. It is impossible to check his record carefully, but it is known that in the three years of his use on the cows herein described many of them had genital disease. In his breeding period, Herd I had 11 abortions, 1 retained placenta with prolapse of the uterus, 6 cases of metritis, and 8 of sterility. Thus he bred nineteen cows with a history of genital disease. He was killed for beef in May 1923, and no examination of his genitals or semen was made.

Bull "B", born in 1919, was purchased from a large breeding establishment at 7 months of age. A few cows from Herd I only were bred to him late in 1921. In 1922 he was pastured with about twenty of the heifers and cows. His services did not extend to any other herd until April 1923, when cows were brought to him from Herd II. His breeding service was moderate until the spring of 1924 when possibly he was overworked. In March 1924 sixteen or more services were required of him, and in May and June he was bred to twenty-four or more cows each month. Often three cows were bred on the same day. In Herd I the following cases of genital disease occurred in cows that he bred: 17 abortions, 1 retained placenta, 7 metritis, and 9 sterility. This involved twenty-four cows, four of which had previously had trouble after service to bull "A", and eleven of which had never been bred to any other bull. This sire left the herd in March 1925. He passed six herd blood tests that were entirely negative and reacted slightly at 1:60 Jan. 6, 1925. In January 1925 a sample of semen taken from the vagina of a heifer was injected into a guinea pig, and the pig died. The injection was repeated with the same result. Feb. 2, 1925, a sample of semen was procured by means of massage of the seminal vesicles. Material from one-half was injected into a guinea pig with negative results. Material from the other half was injected into another guinea pig, which developed definite lesions of *Bact. abortum*.

DEVELOPMENT OF BACT. ABORTUM INFECTION IN THE TWO HERDS THAT WERE BRED TO BULLS "A" AND "B"

In April 1923 bull "B" came in breeding contact with Herd II consisting of 30 cows in which there had been four abortions in August and September 1922. Thorough laboratory examinations of three of these four cases failed to reveal *Bact. abortum* infection except in the uterine exudate of one cow, number 100. The herd agglutination at this time was also negative. In 1923 there were two abortions. A herd agglutination test in January 1923 revealed one cow positive to the agglutination test, and she was immediately killed. A second test in June revealed another positive cow, and a third test in November revealed six. From the fall of 1923 until the fall of 1924 numerous cows were bred to bull "B" from this herd. A herd agglutination test in June 1924 gave twenty-one reactors. Seven abortions occurred during the year, one in January, and one in March; the others were from August to the end of the year. Laboratory examinations revealed *Bact. abortum* in each case.

Beginning with the fall of 1922, when evidence of the infection was limited to the uterine exudate of cow No. 100, the herd developed into a highly infected group in 1924-25. During the latter part of this period bull "B" was in frequent breeding contact with infected cows. The cows were led to the yard at the farm where Herd I was kept and immediately returned to the farm of Herd II. In no case were they bred within less than two months after

parturition. This breeding service constituted the only point of contact between the cows of the two herds.

It is interesting to note that the first evidence of *Bact. abortum* infection in Herd I was like that observed in Herd II; namely, positive uterine exudate, with this difference, that it came from cows that had not aborted. In Herd I the first positive uterine exudate was from cow number 20, age 6 years, in March 1923; she did not abort, but suffered from a fatal metritis alone. It also should be noted that bull "A" had been in similar contact with Herd II, and had bred on January 30, March 12, and April 1, 1922, cow number 100 that gave a positive uterine exudate after abortion in the fall of 1922. He then bred cow number 20 (Herd I) on March 28, April 20, May 11, and June 3, 1922. The second evidence of *Bact. abortum* infection in Herd I was in March 1924 in the form of a positive agglutination test at 1:20 in cow number 31, and she died in a few days of acute metritis. In March 1924 cow number 24 and cow number 27 gave *Bact. abortum* in the uterine exudate; cow number 24 had a live calf followed by metritis and cow number 27 had a dead calf at term. In May and September following this laboratory examination, both cows were entirely negative to the agglutination test. The fifth cow to become positive was cow number 32. In April 1924 she had a slight metritis after delivery of a live calf, and *Bact. abortum* was found in the uterine exudate. Her blood was negative in the following May, September, and December. The sixth positive cow was number 34 that aborted at 7 months October 12, 1924 while at pasture with the entire herd. This abortion occurred 19 months after the first positive evidence of *Bact. abortum* infection in the herd. She was the first cow with a positive blood reaction to abort. *Bact. abortum* was found in the placenta, fetus, and milk. This animal offered an abundant opportunity for the spread of the infection that soon followed.

TABLE 3—GENITAL DISEASE IN EACH YEAR

	1920	1921	1922	1923	1924	Three months, 1925	Total
Abortions	3	3	5	4	11	3	29
Metritis alone	2	2	2	3	6	1	16
Retained placenta without abortion	.....	.....	1	.....	1	.....	2
Retained placenta with abortion	.....	.....	.....	2	5	2	9
Sterility	1	4	3	5	4	.....	17
Dystocia	.....	.....	1	2	4	.....	7
Prolapsed uterus	.....	.....	2	.....	.....	.....	2
Placentae removed	.....	.....	1	2	6	2	11
Died of genital disease	.....	1	2	1	1	.....	5
Killed as useless for breeding	2	.....	.....	.....	2	3	7
*Total number of cows having genital disease	5	6	11	11	19	5	39
Per cent of herd affected yearly (average of 30 cows)	16.6	20	36.6	36.6	63.3	.....	.....

* Apparent inconsistencies in these totals are due to the fact that some cows received two diagnoses.

Thirty-nine animals, or 55.7 per cent of the 70 cows that have been in this herd have suffered from genital disease at some time during the years 1920–25. In a group of forty, from which we have records of the agglutination test of the blood, 75 per cent have had some form of genital disease.

The diagnoses are made upon a clinical basis. *Abortion* means that the pregnancy terminated in the expulsion of a dead fetus; *metritis* is used when there was a marked inflammation of the uterus following the delivery of a normal calf and prompt expulsion of the afterbirth; *retained placenta* includes only those that required manual removal after the delivery of a normal calf. When a female required three or more services to become pregnant she was diagnosed as *sterile*.

ABORTION

Twenty-four cows aborted from 1920 to March 1925. Five aborted twice making a total of twenty-nine abortions. Ten of these occurred at or near the end of gestation. Thus 34.2 per cent of the total group of seventy breeding animals aborted during five and one-fourth years. Table 3 shows the annual distribution. The approximate annual rate was 10 per cent in 1920; 16.6 per cent in 1922; 13.3 per cent in 1923; 30 per cent in 1924; and 10 per cent in the first three months of 1925. The rate for the preceding ten years was 10 per cent or less. Further observations show that sixteen abortions, 55.1 per cent, followed previous genital disease; these were Cases 3, 6, 11 (twice), 12, 13, 18, 19, 22 (twice), 23, 24, 26, 27 (twice), and 29. Thirteen had no history of prior trouble; these were Cases 1, 5, 9, 10, 14, 21, 23, 24, 34, 36, 37, 38, and 39. Eight followed sterility, 28.1 per cent. Three were directly followed by sterility, 10.3 per cent.

METRITIS ALONE

Fourteen cows, or 20 per cent of the breeding animals, showed evidence of metritis without retained placenta or abortion. Others undoubtedly had a metritis that was not discovered. All of the metritis cases were diagnosed within the first two weeks after an apparently normal parturition. One had aborted the previous year, and one aborted the year following a normal parturition with metritis.

STERILITY

Seventeen cows, or 24.3 per cent of the breeding animals, fell within the definition of sterility and required three or more breedings for conception. As pointed out in the discussion under abortion, eight cows, 47 per cent of the sterility group, aborted in the following pregnancy. Three, or 17.6 per cent of the sterile cows, aborted at the parturition preceding sterility. In no case of sterility was there found evidence of the presence of *Bact. abortum*, although two of the group revealed this organism in their uterine exudates at the following parturition.

RETAINED PLACENTA

The placenta was retained after a live calf at term in only two cows. In one, Case 10, an abortion had occurred three years previously followed by three healthy calves. The other, Case 18, became sterile, and finally conceived and aborted with a second retained placenta. Eight other cows retained their placentae after abortion. This gives a total of eleven. Fifty-four per cent were in cows negative to various tests for *Bact. abortum.*

DYSTOCIA

Six cows, or 8.5 per cent of the total, suffered from dystocia, and it is possible that several others may have received slight aid from attendants. All six developed a severe metritis which was a probable cause of the dystocia; their numbers are 9, 18, 22 (twice), 23, 25, and 27. The fetus was alive in only one, but *Bact. abortum* was found in only two—Nos. 27 and 22.

PROLAPSE OF THE UTERUS

Two cows suffered from prolapse of the uterus, and one from intussusception of the uterus. The first prolapse (Case 18) occurred thirty-six hours after parturition with retained placenta; this cow was sterile for a year and then aborted at six months. The second prolapse (Case 19) occurred with an abortion at six months; she died in a few days from septic metritis associated with a slight traumatic gastritis. The intussusception (Case 26) occurred with a six-months abortion; this cow was sterile for nine months, followed by a normal parturition.

Bact. abortum was not associated with any of them, but a mould was found in the two abortions.

NATURE OF THE GENITAL DISEASES

Of the thirty-nine cases discussed, eight (Nos. 10, 11, 18, 22, 23, 24, 27, and 29), 20.5 per cent, seem to have a chronic metritis with or without abortion or retained placenta in two or more successive years. A chronic disease is also suggested by the fact that eight other cases (Nos. 3, 6, 7, 12, 13, 17, 26, and 28), 20.5 per cent, suffered from genital disease in successive years.

With our limited bacteriological data it is impossible to state what part *Bact. abortum* has played in the genital disease of this herd, but an attempt to trace what happened after its presence was detected is interesting. There is little evidence that it was associated with the genital disease occurring before March 1924, with the possible exception of the positive metritis exudate found in No. 20 in March 1923, and she died at once.

Based on the evidence obtained by the blood tests and other material examined, the herd was classed as practically negative until March 1924. The first positive evidence that we obtained was a positive uterine exudate in March 1923; and the next was a

blood reaction of 1:120 in March 1924. In each case the animal died shortly after the sample was taken. In March and April 1924 *Bact. abortum* was obtained from the uterine exudates of Cases 24, 27, and 32, although their blood and all other material were negative. Two months later a blood test of the herd showed all cows to be completely negative in all dilutions. Another herd test on September 19, 1924, however, revealed three more reactors to the agglutination test. Nos. 34, 36, and a heifer were complete at 1:80. Five days later the blood of No. 36 was complete at 1:40. She has shown no evidence of infection since, except that a guinea pig showed *Bact. abortum* lesions when injected with her milk taken at the time of parturition.

On October 10, 1924, No. 34 aborted at pasture when she was out with practically the entire herd. Her placenta was expelled at once and lost, but the fetus, uterine exudate, and milk were positive to *Bact. abortum*. She was isolated for a few weeks and sold as unprofitable. Of the herd of pregnant cows and heifers at pasture with No. 34, ten aborted between December 10, 1924, and February 11, 1925, while two others, No. 35 and a heifer, calved prematurely. The ten abortions following No. 34 occurred in the following order: Nos. 36, 11, 37, 38, 27, 23, 22, 39, 24, and 29. In all these there was some evidence of *Bact. abortum* infection. In Nos. 11, 22, 24, 27, 29, 38, and 39, *Bact. abortum* was present in the uterine exudates and placentae and in the feti and milk. In the two that calved prematurely, and in Nos. 23 and 37, there was simply the positive reaction to the agglutination test.

A careful analysis of the histories of the above ten cows shows that six (Nos. 11, 22, 23, 24, 27, and 29), or 60 per cent, had suffered previously from some form of genital disease. An abortion in cows with such histories is not infrequent when *Bact. abortum* is absent. In addition, Nos. 34, 38, and 39 have imperfect breeding records.

On examination of the uterus of No. 34 a few hours after abortion adventitious placentae were noted over the bases of the uterine horns, possibly indicating that she had suffered from metritis at the previous pregnancy. No. 38 is a 21 months-old heifer whose genital tract was imperfect as shown by an examination before breeding. No. 39 was temporarily sterile in 1923. No. 37 had a good history and her genitals were never examined. No. 35 had a good history except that she required two services to become pregnant.

The tendency of the genital infection in this herd to become chronic is evident. Until March 1924 the average yearly abortion rate was 13.3 per cent, but other symptoms of genital disease were plentiful. During the year following March 1924, *Bact. abortum* infection was present and the abortion rate was approximately 35 per cent. This evidence supports the general belief, that when *Bact. abortum* infection enters a previously negative herd, the rate of abortion increases.

Bacteriological Work

While the bacteriological work is somewhat limited it tends to show that *Bact. abortum* was not the only organism that caused trouble. The following brief summary is an attempt to show the importance of the various organisms in the different types of genital disease in this herd.

Abortion. In ten abortions (Cases 11, 22, 24, 27 (two), 29, 34, 36, 38, and 39), or 34.4 per cent of all abortions, *Bact. abortum* was found in some of the material examined. Also in two others (Cases 23 and 37) the blood was positive to the agglutination test, but other material was not examined. This makes a total of 41.3 per cent of the abortions that were associated with *Bact. abortum* infection. In eight, or 27.3 per cent of the abortions, *Bact. abortum* was the only organism found. Other organisms were isolated from fresh material obtained from cases of abortion as follows:

Streptococcus, 17.2 per cent (Cases 12, 18, 19, 24, and 26).
B. coli 13.7 per cent (Cases 9, 11, 18, and 24).
* Mould, 10.3 per cent (Cases 11, 19, and 26).
Staphylococcus, 6.8 per cent (Cases 11, and 19).

In Cases 23 and 36, no bacteria were found, and feti from Cases 24, 29, and 38 were sterile by culture and guinea pig inoculation. Twenty-seven and one-half per cent of the cases of abortion were not examined.

Metritis. Of the fourteen cases suffering from metritis, only seven received bacteriological examination of the uterine exudate. *Streptococcus hemolyticus* was present in Cases 7, 28, 29, and 31. A diplococcus was found in No. 32. *Bact. abortum* was present in Cases 20, 24, and 32, and a slightly positive reaction to the agglutination test occurred in Case 31. *B. pyogenes* was found in Case 24 and *B. coli* in No. 28.

Retained Placenta. There were but two cases of retained placenta and one of these was negative to *Bact. abortum* on guinea pig inoculation with placental extract, but did not receive a complete bacteriological examination. The other, Case 10, yielded a streptococcus and a staphylococcus from culture of the placenta. There was no evidence of *Bact. abortum* infection in either case.

This bacteriological work is incomplete because material from the uterus was not examined at the time of the acute metritis. In many cases, also, where material was obtained there was only enough time to search for *Bact. abortum.*

Treatment

This herd received the routine treatment applied to cows suffering from diseases of the genital organs. The aim of the treatment of all cases of acute metritis was to return the uterus to normal size as soon as possible after parturition, and to leave the entire genital tract as free from infection as possible at the end of the

* These cases form part of a report by Gilman and Birch in the Cornell Veterinarian, April, 1925, p. 81.

puerperal period. In handling cases that had been bred repeatedly and failed to conceive the same principle of disinfection of the uterus, cervix, and vagina was applied. In addition the retained *corpora lutea* were removed from those cows where estrum failed to appear. The ovaries were massaged, and large cysts ruptured in some of the sterility cases where estrum did appear, but as a rule in these the ovaries were not treated.

To accomplish the desired results in metritis, hot saline douching and removal of all debris from the uterus were used in nearly every case. If the placenta was retained it was usually removed forty-eight or more hours after parturition. The uterus, having been stimulated to contraction and freed of exudate, was filled with liquid petrolatum; and this was repeated as often as necessary to control the infection. Lugol's solution (5 cc. to 500 cc. of liquid petrolatum), or iodoform and bismuth subnitrate one-half ounce of each to a pint of liquid petrolatum, were found excellent as a protectant for the uterine mucosa in acute or subacute metritis. Pituitrin in 2 cc. doses daily seemed to hasten involution of the uterus. In sterility the uterus was disinfected with 1 to 2 per cent aqueous solution of Lugol's, after the mucus and pus had been removed with a saline douche. The cervix was a frequent seat of inflammation. This was relieved by swabbing with full strength Lugol's solution or other preparations containing iodine. An ounce or two of a non-irritant antiseptic was left in the uterus after the above treatment in an attempt to control the infection in the uterus, cervix, and vagina. For this, 0.5 to 2 per cent Lugol's solution in liquid petrolatum, or a 1:500 solution of acriflavine in glycerine proved useful.

The control of the disease by isolation in box stalls at the time of calving, or aborting, was attempted and carried out better than in the average herd. However, many abortions occurred unexpectedly at pasture or in the main barn. Many other cows known to be discharging abnormal amounts of the so called "lochia"—really metritis exudate—were placed with the main herd too soon after parturition. We feel that about all we were able to accomplish by isolation was to keep the stable floors of the main barn reasonably free from pathogenic organisms. To accomplish this the severe acute metritis cases were isolated while under treatment.

The herd was as well isolated as possible, except that cows from Herd II were bred at this farm by the herd sires "A" and "B." Herd II was highly infected with *Bact. abortum,* but had a low abortion rate until the fall of 1924.

RESULTS OF TREATMENT

Due to the fact that many cows were sold from the herd as breeders, complete data regarding the results of treatment are lacking. Thirty of the thirty-nine cases described were treated for some form of genital disorder.

Abortion. Among the thirty cases that were treated eighteen abortions occurred in sixteen cows and were treated near the time

of parturition. Five, or 27.7 per cent of those treated for abortion, delivered live calves at term at the next pregnancy. One of these was sterile for a time after the treatment for abortion, and another developed severe metritis after an apparently normal parturition. Two, or 11.1 per cent of the abortion group, aborted during the next pregnancy. Two died from the perimetritis following an early abortion—11.1 per cent. Three more were killed shortly after aborting since their probable value in a dairy herd was less than their value as beef, thus making a total of five, or 27.7 per cent of this group, lost because of this type of genital disease. The termination of the others in this group is unknown.

Eleven other abortions received no attention at the time of abortion or before they were bred again. Three, or 27.2 per cent, of these calved normally at the following parturition; but one gave premature calves each year following and aborted three years later. Two, or 18.1 per cent, were killed as hopeless breeders, while the termination of the remainder is unknown. This might seem to indicate that treatment had no effect upon the percentage of cows saved as breeders, but this is due to the fact that the cases treated were accompanied by either prolapse of the uterus, dropsy of the amnion, emphysema of the fetus, retention of the fetal membranes, or severe metritis. Those not treated were abortions with no evidence of severe metritis.

Metritis. Three, or 23 per cent of the thirteen cases treated for metritis without retained placenta or abortion, calved normally at the next parturition. Two, or 15.4 per cent, aborted during their next pregnancy. Three, or 23 per cent, died principally because of septic metritis, and two others became useless as breeders following the metritis. Thus 38.4 per cent of those treated for acute metritis were lost as breeders. The termination in three cases is unknown.

Removal of Placentae. Eleven placentas were removed, nine of which were retained accompanying an abortion. Twenty-seven per cent of these calved normally at the following parturition. Two, or 18.1 per cent, were temporarily sterile after the placenta was removed. Two aborted at the following parturition, and one of these was then killed as beef. The results in the remainder of the group are unknown.

Sterility. Ten of the sterility cases were treated. Seven, or 70 per cent of them, calved normally at the following pregnancy. Two, or 20 per cent, aborted when they did become pregnant, and the other one remained sterile and was sterile two different years, calving normally each time after her uterus and cervix were disinfected.

Eleven cases of sterility were noted in ten other cows, but no treatment was given. They were bred from three to seven times each, and all finally conceived. Three, 27.2 per cent, calved normally, but one of these after calving required two services to become pregnant the second time and then suffered from a severe metritis with retained placenta. Two others, thus making a total

of 45.5 per cent, produced normal calves at term, but these two died in a few days of septic metritis. Six, or 54.5 per cent, aborted during the parturition immediately following untreated sterility. Two of these aborters were killed as useless breeders. Thus it is seen that 70 per cent of the treated sterility cases were saved as breeders, while 27.2 per cent of those untreated remained in the herd.

SUMMARY OF TREATMENT

Of the thirty cows treated, 90 per cent were attended because of acute metritis either with or without abortion and retained placenta. Thirty per cent of these treated calved normally at the following parturition, but in two cases metritis appeared after the normal calving. Of this group of twenty-seven cows, 16.6 per cent aborted at the next parturition after treatment, and 36.6 per cent either died or were killed as useless breeders.

In this herd, therefore, when the infection was severe enough to cause an acute metritis at the time of the expulsion of the fetus, it was possible to save the cow as a normal breeder in about thirty per cent of the cases. In these twenty-seven cases the presence of *Bact. abortum* infection was noted in 36.6 per cent. Sterility if treated caused but 30 per cent loss, and if untreated practically ruined 73 per cent of those affected.

GENERAL SUMMARY

1. Observations upon a herd of cattle completely negative to evidence of *Bact. abortum* infection, showed that as high as 36.6 per cent suffered annually from genital diseases.

2. While the herd was negative to *Bact. abortum* infection, abortions frequently occurred in cows that conceived after untreated sterility.

3. Organisms associated with abortions in the herd at the time it was negative were — mould (genus Mucor), colon, and streptococcus.

4. This negative herd became infected with *Bact. abortum* although the only known contact with infected animals was through two bulls which were bred to this herd and an infected herd.

5. The semen of one herd sire caused *Bact. abortum* infection in a guinea pig which was inoculated.

6. One cow, Case 34, aborted at pasture with the entire herd, and was positive to *Bact. abortum* infection. Starting two months later the herd developed a high rate of abortion, all of which was associated with *Bact. abortum.*

7. After this herd became highly infected with *Bact. abortum* the yearly abortion rate increased 21.7 per cent.

8. Positive evidence of infection with *Bact. abortum* was obtained one year earlier by the examination of uterine exudate than by the agglutination test of the blood.

PART II

INDIVIDUAL RECORDS

Case 1, born 1918, daughter of cow described under Case No. 3. Aborted September, 1920, at 6.5 months after one recorded service.

Case 2, born 1917. Calved normally in 1919 from service to bull "A." She conceived again to the same bull, and developed a severe septic metritis after live twins in May 1920. Sold because of disease of the genital organs.

Case 3, born 1907. This cow delivered a live calf each year, but in 1918 three services were required for pregnancy, and live twins were expelled at 267 days. She was then bred seven times with no treatment and aborted a dead calf at term on July 3, 1920. Sold for beef.

Case 4, born 1918. Metritis in May 1920 after delivery of a live calf from service to bull "A." She was sold as a breeder and has since been treated for sterility.

Case 5, born 1919. Bred to bull "A" in 1920. Aborted February 1921 at term. No treatment was given and no further record is available.

Case 6, born 1918. May 17, July 19, and December 1, 1919, bred to bull "A." September 11, 1922, she delivered one dead and one live twin. No treatment. Sold.

Case 7, born 1917. This cow gave birth to two normal calves after one service each year to bull "A." December 30, 1920, bred to bull "A"; January 18, 1921, bred to bull "A"; February 23, 1921, bred to bull "A." December 6, 1921, calved normally. Fifteen days later she developed symptoms of meningitis and died after seven hours. The autopsy revealed meningitis and a chronic circumscribed traumatic peritonitis due to an old perforation of the reticulum with a nail. The uterus contained about a liter of reddish exudate. Cultures from this showed a *Streptococcus hemolyticus.* Guinea pig inoculation failed to reveal evidence of the presence of *Bact. abortum.*

Case 8, born 1919. Calved at term in March, 1922, from service to bull "A." The calf died on the third day and a few days later the cow was treated for metritis. She was later sold as a breeder.

Case 9, born 1920. This heifer conceived from one service to bull "A." Abortion followed at the 7th month in February, 1922. Emphysema of the fetus caused dystocia and she died in a few days from peritonitis and perimetritis. The fetus yielded a three-sugar colon organism on culture.

The following thirty cases have a record of blood agglutination tests for the presence of *Bacterium abortum* anti-bodies in addition to other bacteriological and clinical information.

Case 10, born 1918. She aborted in August 1920, at 8 months after service to bull "A," and required treatment for slight metritis and vaginitis. In 1921 she was bred three times to bull "A,"

and in 1922 four times to bull "B," before conceiving. She calved normally in 1922 and 1923. In 1923 she was bred to bull "B" in May and June, calved February 28, 1924, and had a retained placenta. This was removed on the third day, but a rather severe metritis and cervicitis followed. She bred again to bull "B" on May 28, 1924, and delivered a calf at term. A culture of the placenta in 1924 yielded a streptococcus and a staphylococcus.

Case 11, born 1917. She calved in March 1920 after four services to bull "A" with no treatment for sterility, and aborted January 1, 1921, after three services to the same bull; the placenta was not retained and no treatment was given. She was again bred to this bull on March 8, 1921, and calved normally December 5, 1921, at 272 days; but the calf developed pneumonia and scours. She was bred March 13, 1922, to bull "B" and expelled a weak calf on January 1, 1923, at 263 days. Following this, without treatment or examination of the genitals, she calved normally at 282 days on December 23, 1923. In 1924 she was bred in March and April to bull "B" and aborted at 229 days; the placenta was not retained. The uterus was now treated for the first time; it received Bipp after a saline douche, and pituitrin was injected. Recovery followed promptly. December 12, 1924, the milk, fetus, and uterine exudate revealed *Bact. abortum* on guinea pig inoculation. The fetal lung also yielded on culture a mould, staphylococci, and *B. coli.*

Case 12, born 1920. Bred in April, May, June, and December, 1921, to bull "A." Aborted in July, 1922, at 6 months while at pasture with other pregnant cattle. Treatment for metritis followed, and a retained corpus luteum was removed in January, 1923. Bred to bull "B" a few days later, and delivered normal twins November 7, 1923. Bred once again to bull "B" and sold in the fall of 1924 while in advanced pregnancy. *Laboratory examination:* August 1922, milk negative to *Bact. abortum;* cervical pus contains *Streptococcus alpha.*

Case 13, born September 1919, daughter of cow described under Case 26. Bred to bull "A" December 1920, January 1921, and February 1291. She aborted at term and no treatment was given. Bred to bull "A" and calved at term, one year after she aborted. Bred to bulls "A" and "B," but failed to conceive and was treated for sterility March 23, 1923. Conceived at first service to "B" and calved normally May 1924. Bred to "B" but was returned for sterility treatment June 1924. Nodular vaginitis, slight cervicitis and slight metritis were present. Bred in August to "B" and sold in December, 1924, while definitely pregnant from this service.

Case 14, born 1918, daughter of cow described under Case 26. Bred twice to bull "A" in 1920 and calved December 1920. This calf is described as Case 24. Bred twice to bull "A" and aborted at term in the fall of 1922. No treatment was given, and after one service to bull "B" she calved normally in 1923. Bred once to bull "B" March 1924, and calved normally in December 1924.

Case 15, born 1920, daughter of cow described as Case 19. Bred to bull "A" and February 1922 gave birth to a weak calf (described as Case 28) and suffered rather severe metritis. This was treated with a saline douche followed by a 1 per cent Lugol's solution in liquid petrolatum, which was left in the uterus. Bred to bull "B" 1923 and 1924 and calved normally.

Case 16, born 1919. Bred to bull "A" and calved normally in the fall of 1921. Bred to bull "A" repeatedly until June 1922. She then showed nodular vaginitis as the principal cause of sterility. This was treated repeatedly with a 0.5 per cent Lugol's solution, and she conceived promptly and has calved normally each year since. She frequently shows estrum and is bred while pregnant, but calves from the first service.

Case 17, born 1920. Supposedly bred to bull "B" at pasture during summer of 1922. December 1922, treated for sterility due to cystic ovaries, cervicitis, and severe nodular venereal disease. Bred to "B" and calved at term October 29, 1923. Metritis treated November 7, 1923, by placing a pint of 1 per cent Lugol's solution in liquid petrolatum in the uterus. Fall of 1924 gave birth to normal twins and was sold at once as a breeder.

Case 18, born 1909, dam of cows described as Cases 19 and 26. This cow calved normally each year. Bred to bull "A" July 9, 1921, and September 17, 1921. Calved at term, July 1922, and after the placenta had been retained thirty-six hours the uterus was prolapsed. Recovery from the acute metritis was prompt, but chronic cervicitis and metritis that remained caused sterility. She was bred to both bulls, and after treatment for sterility was bred to bull "B" in September 1923. Hydrops of the amnion and allantois with abortion and dystocia at 6 months required treatment on March 7, 1924. The membranes contained many gallons of fluid, and were very edematous. The placenta was of a diffuse type over the entire uterus. The membranes were removed during the next forty-eight hours. Bipp was kept in the uterus, and pituitrin was injected in 3 mil doses daily for a week. Patient remained in good condition, and when killed for beef two weeks later the genital tract showed principally a thickening of the uterine wall. *Laboratory examination:* July 1922 and March 11, 1924, placenta and milk were both negative to *Bact. abortum* infection. March 7, 1924, placenta yielded beta (hemolytic) streptococcus, uterine exudate, and fetus negative to *Bact. abortum;* culture of fetus showed *B. coli* from all parts. March 18, 1924, uterine exudate gave a hemolytic streptococcus on culture after tract was removed from cow, and guinea pig injection of same exudate was negative to *Bact. abortum.*

Case 19, born 1914, daughter of cow described as Case 18. Four services required for her first pregnancy, but calved normally at three years of age and has given a live calf at each pregnancy since. Bred to "A" in May and June 1921 and calved normally in March 1922. Bred to "B" in May and June 1922 and aborted at sixth month of pregnancy with prolapse of the uterus. Severe

metritis was present and the cow died six weeks later from metritis, perimetritis and slight traumatic gastritis. *Laboratory examination:* blood negative October 26, 1922, December 19, 1922, and January 4, 1923. Milk negative by guinea pig injection December 19, 1922. Fetus yielded a pure culture of mould (genus Mucor) from the lungs and all parts of the digestive tract. The fetus and placenta were negative to *Bact. abortum* infection. Post mortem culture of the uterus gave streptococcus and staphylococcus.

Case 20, born 1917. First three parturitions were normal. Sterility occurred in 1922 and was not treated. Bred to bull "A" in March, April, May and June, 1922, and calved in an apparently normal manner March 12, 1923. March 22, 1923, had severe septic metritis and showed the symptoms of parturient paresis. Several ounces of dark reddish exudate were siphoned from the uterus. The udder was inflated and stimulants were administered. The cow regained a standing position in a few hours, but the paralysis and collapse returned and it was necessary to inflate the udder several times in order to keep the patient on her feet. The uterus gradually returned to normal size, but the cow died of general septicema from the metritis. A chronic circumscribed peritonitis with traumatic gastritis was also present. *Laboratory examinations:* blood, partial agglutination at 1:40 March 23, 1923; milk negative to *Bact. abortum* infection March 27, 1923; metritis exudate not cultured but was reported positive to *Bact. abortum* infection by guinea pig inoculation March 23, 1923. This was the first evidence of the presence of *Bact. abortum* infection in this herd and no more positive evidence was obtained until about one year later.

Case 21, born 1921. Bred to bull "B" and aborted in January 1923 at the 278th day of pregnancy. Sold at once for breeding purposes.

Case 22, born 1920. Heifer was bred several times to bull "B" and in February 1923 received treatment for sterility. A persistence of the embryologic structures, Muellerian ducts, left the heifer with a band posterior to the external os of the cervix. The uterus and cervix were disinfected. Bred to "B" and aborted at term in February 1924. Slight metritis followed. The band at the external os was severed in March 1924, and the cervicitis and metritis treated repeatedly during the next six weeks. Bred May 1, 1924, to bull "B" and aborted with dystocia eight months later. The placenta was expelled in ten hours with the aid of pituitrin subcutaneously in 3 mil doses at three hour intervals. Study of this placenta showed that the non-gravid chorion consisted of a non-placental pouch-like sac extending into the non-gravid horn for a distance of 6 inches. *Laboratory examination:* the negative blood agglutination is the only information regarding the first abortion. After the second abortion, December 30, 1924, it was found that the chorionic cells were filled with *Bact. abortum.* Placenta and metritis exudate were also positive, while the fetus and milk were negative to *Bact. abortum* infection by guinea pig

injection. Fetus not cultured. There is ample clinical evidence that this cow might have aborted regardless of the entrance of *Bact. abortum.* One uterine horn did not function in placental attachment and severe cervicitis existed before she was bred and after aborting.

Case 23, born 1918. Normal parturition occurred in 1920 and 1921 after one service each year to bull "A." Bred to bull "A" in April 1922 and aborted with dystocia and retained placenta at 276 days. The placenta was removed and severe metritis was treated a few times. Bred once to bull "B" in 1923 and calved normally in February 1924. Bred May and June 1924 to bull "B" and aborted at 192 days in December 1924. Sold just before aborting and no material except the blood shortly before abortion was obtained. Blood, placenta, and fetus at time of first abortion failed to show evidence of *Bact. abortum* infection, and cultures of the fetus were sterile.

Case 24, born December 1920. Bred to bull "B" April 1922, and aborted at term February 1923. Severe metritis required two treatments. Bred to bull "B" and delivered a live calf at term March 1924. Severe metritis was treated twice and the cow was bred to bull "B" May 10, 1924. Aborted at 8.5 months on January 26, 1925. Placenta was easily removed immediately after the abortion occurred. Metritis not severe. *Laboratory examination:* milk negative to *Bact. abortum,* infection on guinea pig injection of samples procured February 1923, April 1924, and September 1924. February 7, 1923, fetus and metritis exudate negative to *Bact. abortum,* but culture of metritis exudate March 24, 1924, gave hemolytic streptococcus, and *B. coli.* A guinea pig injected with the placenta developed lesions typical of *Bact. abortum* infection; cultures of the metritis exudate April 2, 1924, gave *B. pyogenes.* Smears made from the chorion January 26, 1925, showed *Bact. abortum* in the epithelial cells; and the fetus, placenta, and milk were positive to *Bact. abortum* infection.

Case 25, born 1921. Dystocia occurred in May 1923 due to a fibrous band similar to the one described in Case 22. Removal of this band at external os relieved the dystocia, but slight metritis was treated eleven days later. Sold as pregnant fall of 1923.

Case 26, born June 1915, daughter of cow described as Case 18. This cow gave a normal calf each year after one or two services to bull "A". Calved at 282 days in 1922 and developed parturient paresis. Made a prompt recovery and the uterus was not examined. Bred once to bull "A" and calved normally at 281 days in March 1923. Bred to bull "B" on April 14 and May 6, 1923, and aborted with retained placenta and intussusception of the uterus November 1923. Placenta removed and intussuscepted gravid horn replaced six hours after abortion. The severe metritis and cervicitis were treated principally with antiseptics in liquid petrolatum. These were placed in the uterus frequently. Bred a few times to bull "B" after her genitals returned to normal; but sterility, due to chronic metritis, cervicitis and cystic

ovaries, was treated several times. She then became pregnant by bull "B" and calved normally early in 1925. *Laboratory examination:* besides a negative reaction to the agglutination test at the time of abortion and all other times, the milk, fetus, and placenta taken on the day of abortion were negative to *Bact. abortum.* Culture of the fetus showed a mould (genus Mucor). Culture of the cervical mucopus on January 2, 1924, gave a streptococcus and an unidentified rod.

Case 27, born March 1914, a valuable A. R. O. cow. Aborted at term at 21 months of age in 1915. Calved normally each year, after one service by bull "A" in 1916 and 1917. Bred once to bull "A" and aborted at six months in 1918. Bred to "A" three times and calved normally in 1919. Calved normally in 1920 and 1921 after one service each year. Bred twice in 1922 to bull "A" and calved normally with twins. Bred four times in 1923 to bull "B" with no sterility treatment. Aborted at term with dystocia due to fetal ascites March 24, 1924. The placenta was very friable and edematous and the chorion was completely covered with adventitious placentae. The membranes were easily removable at once and the uterus was filled with Bipp. Pituitrin was injected repeatedly. A chronic metritis remained after the metritis discharges ceased. The cow was bred more than once to bull "B" and expelled a 4 months' fetus and all the fetal membranes on December 24, 1924. The placenta was completely covered with adventitious placentae at this abortion. Cow sold as beef because her genital tract was worn out at 11 years of age. *Laboratory examination:* blood reactions negative before, at the time of, and after the abortion in March 1924, but positive at the time of the second abortion in December 1924. In March 1924, the milk was negative to *Bact. abortum* infection, but the placenta and exudate from the uterus were positive to guinea pig injections. In December 1924 the milk, uterine exudate, and all parts of the fetus were positive to *Bact. abortum* infection. Culture of all parts of the fetus also showed *Bact. abortum.* Microscopical examination of smears of the placenta showed no *Bact. abortum* organisms, and there were no gross lesions of *Bact. abortum.*

Case 28, born 1922, daughter of cow described as Case 15. Heifer suffered from calf scours and pneumonia during the first six weeks of her life and her dam had metritis at that time. Bred once to bull "B" and calved at term with slight dystocia February 1, 1924. Metritis and cervicitis required several treatments before the heifer was safe for service. Repeated breeding to bull "B" over a period of a year, followed by slaughter for sterility, ended this case. *Laboratory examination:* culture of vaginal and cervical mucus on March 7, 1924, showed streptococcus, *B. coli,* and an unidentified gram positive rod.

Case 29, born 1922, daughter of cow described as Case 31. Bred once to bull "B" and calved at term March 6, 1924. March 13, 1924, severe metritis treated by saline douche and 1 per cent Lugol's in liquid petrolatum. Four days later gave a saline douche and left Bipp in the uterus. Sixteen days later the cervix and

vagina were disinfected, but uterus not entered. More treatment was to have been given, but the cow was bred to bull "B" on May 5, 1922. Two other cows were bred to "B" the same day. A macerated fetus with the fetal membranes wrapped about it was aborted on February 16, 1925. This fetus had apparently been dead for some weeks. Cow was killed as beef a few days later. *Laboratory examination:* uterine exudate taken in March 1924 gave a hemolytic streptococcus on culture. Milk and uterine exudate at this time were negative to *Bact. abortum* infection. Gross and microscopical examination of the placenta failed to show *Bact. abortum* infection February 16, 1925. Uterine exudate injected into a guinea pig caused *Bact. abortum* lesions in the guinea pig. Fetus and milk examined by guinea pig injections were negative, and cultures of the fetus were sterile.

Case 30, born 1920, daughter of cow described as Case 19. Calved normally in 1923 after being pasture bred to bull "B". Bred to "B" and calved normally March 8, 1924. March 11, 1924, metritis was treated by inserting one-half pint of liquid petrolatum into the uterine cavity. March 17, gave saline douche and Bipp was left in the uterus; repeated March 27, with a menthol-iodine-glycerine swab of the cervix. Calved normally March 29, 1925, at 280 days after one service to bull "B".

Case 31, born August 1916. This cow was bred by bull "A" for the first four pregnancies. Bred to bull "B" and calved at 278 days in 1923. Bred by bull "B" and calved at term March 12, 1924. Symptoms of parturient paresis developed twenty-four hours later, and on March 17 death occurred from septic metritis and mastitis. Autopsy also revealed a liver abscess and a chronic circumscribed traumatic peritonitis. Treatment with inflation of the udder, stimulants and the usual Bipp and pituitrin for the uterus were of no avail. *Laboratory examination:* March 13, 1924, blood reaction to the agglutination test—partial at 1 : 160, being the first reaction higher than C-40 from samples taken from this herd. The uterine exudate was negative to *Bact. abortum* infection, but on culture it proved to contain a hemolytic streptococcus. Udder and milk showed *Staphylococcus aureus.*

Case 32, born December 1919. Bred to "A" twice in 1921 and calved normally April 27, 1922. Bred once to bull "B", and April 1923 calved normally. Bred June 19, 1923, to bull "B" and calved normally March 28, 1924. Placenta was promptly expelled but cow showed slight metritis. Bred to bull "B" May 31, 1924, and sold in December definitely pregnant. *Laboratory examination:* cultures of the placenta on March 28, 1924, showed diplococci, and a gram positive rod, but both may have been contaminants. Metritis exudate obtained April 2, 1924, caused lesions of *Bact. abortum* when injected into a guinea pig. The milk taken on September 13, 1924, is negative on guinea pig inoculation. The blood of this cow was negative from October 1922 to December 1924.

Case 33, born January 1916. Bred by bull "A" and gave a live calf each year, with twins two years. Last parturition was Jan-

uary 26, 1924, with twins. After this, bred in April, May, and June 1924 to bull "B," and estrum has appeared at regular intervals since last service. Examination for cause of sterility in July 1924 showed adhesions about right ovarian ventricle, and a slightly larger uterine horn on that side. The cervix was slightly thickened and hemorrhagic. A fibrous band, as described in Cases 22 and 25, was found, nearly covering the external os. The band was severed with scissors, the uterus douched with Lugol's solution, and the cervix swabbed with glycerine, iodine, and menthol. Five-tenths per cent Lugol's douche of the vagina three times during the next ten days. Bred at first service to bull "B" and calved normally.

Case 34, born December 1917, daughter of cow described as Case 27. Bred to bull "A" once each year and calved normally from each service for the first three years. Bred once to bull "B" and calved normally October 1923. Bred March 16, 1924, to bull "B" and aborted at the seventh month on October 11, 1924, while at pasture with entire herd. There were no signs of approaching abortion. The uterine placentae, on examination directly after abortion, were observed to be small and closely matted over the uterus, adventitious placentae. She was isolated for five weeks after abortion, but her placentae and uterine exudate were already scattered over the pasture and stable. She was unprofitable either for milk or for breeding purposes and was sold for beef five weeks later. The dam of this cow aborted in March and was positive to *Bact. abortum* by examination of the uterine exudate only. This cow is the first case where there was ample evidence that *Bact. abortum* was associated with an abortion. In the next four months after this abortion three cows and a heifer with fairly good records aborted and six others which had previously suffered from genital disease also aborted. In all of these ten abortions, which followed Case 34, there was some evidence of *Bact. abortum* infection. *Laboratory examination:* October 12, 1924, fetus, milk, and uterine exudate showed *Bact. abortum* by guinea pig injection.

Case 35, born August 1919. Cow was bred once each year and calved normally in 1921, 1922, and 1923. Bred March 19 and April 11, 1924, to bull "B" and expelled a live premature calf at 8.5 months. No material was obtained as cow left the herd just before calving.

Case 36, born December 1920, daughter of cow described as Case 26. Bred to bull "B" once in 1922 and 1923 and calved normally each year. Bred to same bull again March 21, 1924. Two other cows bred the same day, and one was returned for service three weeks later. At the eighth month of pregnancy December 10, 1924, Case 36 aborted twins. The placenta was retained, and the metritis required several treatments. After removal of the placenta the uterus received saline douche and Bipp at five-day intervals, and pituitrin was injected daily for the first week after parturition. December 30, twenty days after abortion, the uterus is normal in size and contains no pus. *Laboratory examination:*

the only evidence of *Bact. abortum* infection in this cow was a blood reaction of complete agglutination at 1:80 on September 19, 1924, and five days later it was complete at 1:40. It has since remained negative. Milk, placenta, uterine exudate, and all parts of the fetus were negative to *Bact. abortum* infection by guinea pig injections from samples taken at time of abortion. A second pig, however, injected with the milk showed lesions of *Bact. abortum* infection.

Case 37, born November 1920, daughter of Case 33. Bred to bull "B" and calved normally after one service in 1922, and sixteen months later, after one recorded service to bull "B," she calved normally. Bred to bull "B" May 29, 1924, and aborted at 205 days in December 1924. Placenta was not retained, and no material, except the blood sample shortly before abortion, was examined.

Case 38, born February 1923. Examination of the genitals of this heifer in April 1924 showed a short right broad ligament, which held the right ovary closely against the right anterior portion of the pelvic wall. Slight nodular vaginitis was present but not treated. Vagina was very narrow. Bred to bull "B" May 8, 1924. (An aged cow from another herd where abortion and other genital disease was plentiful was bred to bull "B" on the same date, and she aborted at 8.5 months and had severe metritis.) Case 38 aborted on December 21, 1924, at 7.5 months and retained the placenta. Placenta was partially expelled at eight hours and the most of it was removed at forty-eight hours. Vagina was so narrow that treatment was difficult. The uterus was frequently filled with either Bipp, or 1 per cent Lugol's in petrolatum. *Laboratory examination:* December 22, 1924, the chorionic cells were choked with *Bact. abortum* organisms. Guinea pig injections of uterine exudate, fetus, and milk showed positive evidence of infection in all three. Cultures of fetus were negative.

Case 39, born March 1921, daughter of cow described as Case 4. Pasture bred to bull "B" and calved apparently normally in May 1923. Bred once in June and twice in July to bull "B," and calved normally April 1924. Bred to same bull June 2, 1924, and possibly later. Aborted at about the seventh month on January 5, 1925, and placenta was retained. This was manually removed twenty-four hours after parturition and Bipp was placed in the uterus. Pituitrin was given once daily for four days, and two weeks later the uterus was douched with saline and received Bipp. Genitals appeared nearly normal on February 2, 1925, when the uterus was douched with saline, and the cervix was swabbed with menthol, iodine, and glycerine. Cow should be treated once more and bred two months later. *Laboratory examination:* chorionic cells were filled with *Bact. abortum* on examination of placenta on January 6, 1925. Also on this date the milk, uterine exudate, placenta, and all parts of the fetus were positive to *Bact. abortum* by guinea pig injection method of diagnosis. Cultures of all parts of fetus abomasum, rectum, lung, and liver showed *Bact. abortum.*

THE CHANNEL OF INVASION OF BACTERIUM ABORTUM WITH SPECIAL REFERENCE TO INGESTION*

R. R. BIRCH AND H. L. GILMAN
Veterinary Experiment Station

It is now an accepted fact among veterinarians that most cattle herds which suffer severely with disease manifested by frequent abortion are infected with *Bact. abortum.* The relation between this organism and disease of the chorion which ultimately determines abortion is well established, but doubt still exists regarding the channels through whch the chorion is invaded. These channels of invasion have a direct and important relation to the time at which infection may take place, and to measures for its control. If the channel of invasion is through the cervical canal only, association of aborters and pregnant cows involves no very great immediate danger for the latter; if invasion regularly occurs through other channels, then the pregnant cow cannot safely associate with spreaders of *Bact. abortum* during the gestation period.

No very satisfactory explanation of the relatively rapid and certain spread of Bang abortion disease through a herd has been recorded, but certain facts point to some important channel of entrance other than the cervical canal. Indiscriminate spread of the infection among females in the same herd bred to different bulls; appearance of serological reactions, followed by abortion, so late in pregnancy as to exclude the probability that the infection causing them usually takes place at or near the time of copulation; frequent abortions observed among pregnant cattle placed in infected herds as well as those which follow introduction of aborters into herds relatively free of genital disease, all point to a type of infection which does not always or usually enter through the cervical canal. *Bact. abortum* is chiefly associated with herd histories which verify the facts just enumerated. In what ways is infection with this organism brought about?

It has been customary among writers on veterinary medicine to ascribe it to ingestion, though experimental proof so far submitted has not been wholly satisfactory. The defects do not consist so much in failures to infect, for these will occur frequently even when organisms of proved virulence are used, but in the fact that many of the experiments resulting in infection have not been free from serious objection. Animals of unknown origin have been used; determinations have been made on a very few animals without the use of controls; cows have been moved a considerable distance to strange surroundings immediately prior to the experiments; they have been fed fetal membranes supposedly containing only *Bact. abortum,* but possibly containing other organisms as well; the influence of the bull has not been considered; non-preg-

* The Cornell Veterinarian, Vol. XV (1925), p. 90.

nant cows have been used; regular serological tests have not been made; and too often the advent of infection has been determined by a limited number of serological tests alone or by the abortion rate alone. Divergent views naturally have developed and much confusion still surrounds this exceedingly important point.

We cannot attempt to review here the many publications that deal with channels of infection of *Bact. abortum*. We can refer but briefly to a few of the more recent ones which have been given prominence because of the rather extensive data involved or because of their authors' long study of genital disease.

Hart and Carpenter[1] record the infection of ten animals in an immunity experiment by large doses consisting of infected milk, macerated parts of fetuses and artificial growths of *Bact. abortum* on solid and liquid media. The infected material was given in drenches and on alfalfa hay. The blood of these animals all developed a high agglutinin titre in a very short time and six of them aborted before the 88th day following infection. One calved too soon for accurate determinations to be made, one calved twenty-seven days following infection and the placenta yielded *Bact. abortum* cultures, while two overcame the infection and were reported as having calved normally. The authors conclude: "The ability to produce abortion in susceptible animals by a single exposure to infectious material given by ingestion was demonstrated. The abortions in these cases occurred from fifty-eight to eighty-eight days after the infection."

Schroeder,[2] Huddleston,[3] Rettger, White, and Chapman,[4] Giltner, Huddleson, and Tweed,[5] Simms and Miller,[6] Connaway,[7] and Carpenter,[8] following experiments involving collectively a considerable number of calves, are substantially agreed that in calves *Bact. abortum* infection by ingestion is not readily brought about, and that it does not usually persist from calfhood to breeding maturity. Carpenter [9] has shown that, following ingestion, the organisms regularly invade the lymph glands adjacent to the head as well as those located variously along the enteron in proximity to parts through which absorption takes place. He has shown also that the organisms do not persist in the lymph glands of calves, but tend to die out when feeding of infected material is discontinued.

Williams,[10] in a recent publication reviewing the paper by Hart and Carpenter, suggests that previous infection and the influence of the bull accounted for the abortions which they attribute to ingestion, during pregnancy, of material containing *Bact. abortum*. Williams, in the same publication, further states: "The veterinarian can with confidence take a group of such heifers and expect 40 per cent of them to abort . . . while a group of adult cows from the same herd or of heifers in first pregnancy from a sexually healthy herd may be placed on a luxuriant diet of Bang's bacillus with indifferent loss."

Rettger, White, and Chapman,[4] following a series of experiments in which infection by ingestion was attempted, conclude: "Infection could not be brought about by repeated oral adminis-

tration of artificial cultures of *Bact. abortum* in milk, or in veterinary capsules, which strongly indicates that the alimentary tract does not offer easy access, if indeed any, to the organism causing the disease.'' These authors caution against regarding their results as final.

Our own work, which is recorded in a previous publication,[11] gave inconclusive results. Heifers fed cultures, both before and after breeding, did not abort, but several of these had previously been given repeated doses of dead cultures in an attempt to immunize. In another immunity experiment, described in the same publication, we administered *Bact. abortum* suspensions in capsule to ten pregnant heifers, six of which previously had received several subcutem inoculations with live cultures. The period of infection was approximately three months, and the capsules were administered three times weekly. No abortions occurred, but there was one premature birth with the membranes containing *Bact. abortum,* and tardy agglutination reactions developed in some of the animals. The experiment did little more than suggest need for further work, in which errors now clearly recognized could be eliminated. We therefore began an experiment in which infection by ingestion was the chief consideration.

Sixteen heifers were assembled for the experiment, nine of which were placed in the infected group and seven of which were used as controls. These animals were divided with careful regard for factors likely to influence breeding. Origin, exposure to previous infection, and number of services required to bring about conception were the chief considerations. Age was given due consideration after the other factors were balanced as best they could be. The animals in the infected group averaged somewhat younger than the controls, but all of the heifers were virgins when the experiment began.

Four of the heifers were born in the experimental barn which then quartered an infected herd. These were 221 and 240 of the control group and 240 and 243 of the infected group. No. 257, infected group, was placed in the barn as a calf and was thus exposed to active infection. No. 215, control, and 212, infected group, were purchased as very young calves, reared in the same herd among reacting cows, removed to non-infected quarters as young heifers, and placed in the experimental barn on Feb. 1, 1924. No. 223, 218, and 235, controls, and 239, 241, 238, and 234, infected group, were born and reared in an infected herd, removed as young heifers to clean quarters, and placed in the experimental barn on Feb. 1, 1924. No. 264, infected group, was purchased about 6 months before the experiment began. She came from a small herd in which no observed abortions had occurred in recent years. Following a completely negative* agglutination test made before she was purchased, she was removed to the experimental barn. No. 214 was reared in a clean herd, and was not in contact with reactors at any period of her life. She was placed in the experimental

* 1–20 minimum dilution used in all these experiments.

barn on Feb. 1, 1924. It will be observed that fourteen of the heifers were exposed to active Bang infection as calves, and that as older heifers they were kept in clean quarters, and later used in the experiment. No. 214 and No. 264 were the only two that had not associated as calves with infected animals.

There had been severe Bang infection in the experimental barn during the winter of 1922–23, but at the beginning of the 1923 pasture season all of the infected cows were removed, and the vacated quarters thoroughly disinfected. Following an interval of approximately nine months, disinfectant was applied a second time, immediately before the experiment began.

The heifers were all bred to one bull, No. 260. He was a pure-bred Holstein, purchased in September, 1923, at the age of nine months. He had not been in service prior to purchase, and the experimental heifers were the only ones bred to him. His genitals were examined at the time of purchase and again when the breeding season began. No semen examinations were made. Monthly blood samples set with *Bact. abortum* antigen failed to agglutinate in any dilution until July 15, when a positive 1-20 was recorded. One month later the titre stood at 1-160, and it has remained consistently high since. However, the reaction appeared 3½ months after the breeding season closed and in no way concerns the pregnancies involved in these experiments. It was due probably to exposure in infected quarters, as he had been allowed to exercise in one of the yards occupied by the heifers in the infected group.

The breeding season began on February 11, 1924, and closed on April 1, an interval of fifty days. In the infected group, eleven copulations were required to produce nine pregnancies, two of the heifers, 234 and 238, each being returned once. In the control group ten copulations were required to produce seven pregnancies. One heifer, No. 215, was returned once and another, No. 214, was returned twice. No individual heifer failed to conceive. Judged by the standard of the relation between the number of copulations and the number of pregnancies produced, the breeding efficiency of the bull in the infected group stands at 82 per cent; in the control group at 70 per cent and in the groups as a unit at 76 per cent. On the whole, no suggestion of serious genital disease appears in this record.

Figure 1 illustrates the plan of the barn and pastures. The heifers were kept in the box stalls in the main part of the barn and allowed to associate freely in the yards and pasture until May 25. On this date a fence was completed, dividing the pasture. The controls were placed in the east portion and the infected group in the west. During the experiment the yards were open at all times except when the bull was allowed to exercise in yard 2, but during the summer the heifers were not placed in the barn except for an hour or two once a week when blood samples were drawn. After Nov. 15, all of the heifers were kept in the barn or yards. Segregation of the two groups could not be considered perfect; nevertheless, the most scrupulous care was exercised to prevent transfer of infection to the control group.

The infection period began on May 26, 1924, and continued until August 27. Strains of *Bact. abortum* of proved virulence were grown in large tubes on slant agar for approximately 48 hours. The growths were washed down with physiological saline, and the suspension was diluted to .75 as measured by the Gates scale. Ten mils of this suspension were allotted to each cow, and this was further diluted with physiological saline until the quantity fed the eleven heifers on a single day was about one liter.

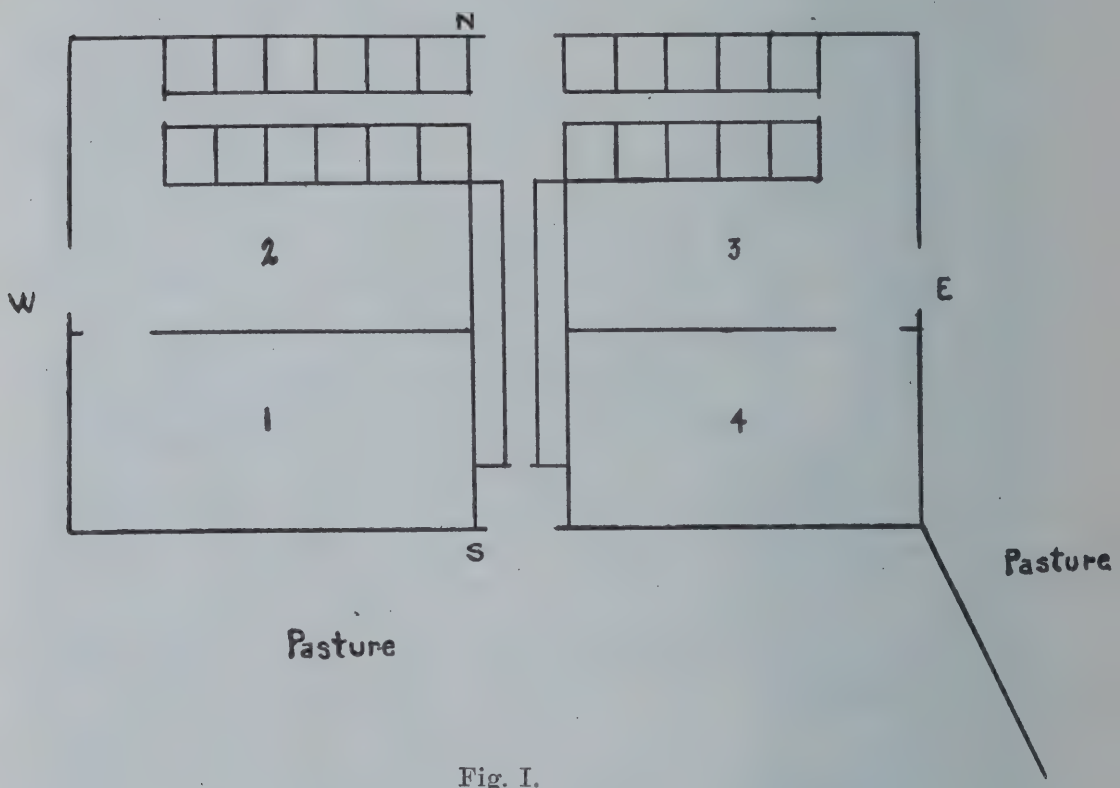


Fig. I.

There was a long feed-trough in yard No. 1. On each feeding date the heifers were assembled in the yard, a quantity of bran and cornmeal was distributed in the trough, and the infectious material was sprinkled over the grain. As a routine, feeding took place each Monday, Wednesday and Friday during the infection period, but there was an occasional delay of one day due to retarded growth of the cultures.

Blood samples from none of the heifers were agglutinating *Bact. abortum* antigen except in very low dilutions at the time the experiment began, and this negative record had prevailed during the several months immediately preceding the experiment. Monthly agglutination tests were continued during the breeding period, but at the beginning of the infection period tests were made weekly. Individual records of these tests are appended.

It will be observed that the heifers were approximately three months in gestation when the infection period began. Past ex-

periences had suggested the desirability of causing infection relatively early in gestation, and Carpenter's work had suggested that inasmuch as in calves at least the glands near the pharynx are most regularly invaded, ingestion of infected material with food might be more certain in its effects than administering it in capsules.

We had for some time regarded it possible that artificial cultivation of *Bact. abortum* might lower its virulence for cattle, and we therefore inoculated a pregnant heifer intravenously, planning to kill her sometime before she aborted, while the cervical seal was still intact, and feed the fetal membranes to the heifers in the infected group. This plan was abandoned because of the unexpectedly prompt response to the artificial cultures as measured by the high agglutinin titre which developed in the blood of several of the heifers. It was evident that the artificial cultures were sufficient to infect.

Tables No. I and II summarize the results of the experiments.

The two groups were fed and cared for alike. The animals were on pasture which late in the season was supplemented and finally replaced by mixed alfalfa and timothy hay. The grain ration for the most part consisted of bran, cornmeal, and ground oats increased as the pasture became less plentiful. Salt was allowed all the animals.

During the experiment all of the heifers with the exception of No. 257 remained in excellent condition except for the fact that there gradually appeared a differentiation between those representing the two groups. Several of the animals in the infected group lost in condition just a little, this being apparent particularly when comparisons were made with the controls.

No. 257 as a calf had been infected intratracheally with material from the trachea of a pneumonic calf and had developed severe pulmonary symptoms. Apparently she had made a complete recovery at the time the experiment began. Prior to that time she had received her feed alone in a box stall, but when fed with the other cattle, being very timid and failing to get her share of the feed, she lost flesh quite rapidly, and was in poor condition at the time the experiment ended. Her record is included in tabulating results, although it changes her status in some degree.

It will be recalled that with two exceptions all the heifers had been exposed as calves to severe *Bact. abortum* infection. The question whether this infection was carried from calfhood to sexual maturity deserves consideration. It probably was not. The negative agglutination record of all the heifers prior to artificial infection, the continued negative record of the controls with regard to blood agglutination, abortion rate, and bacteriological examinations, as well as the readiness with which the heifers conceived, practically rule out previous infection as an explanation of the abortions which occurred in the infected group. In this group the prompt appearance of agglutinins in the blood following artificial infection, the high abortion rate which followed,

TABLE NO. 1 (*Infected Group*)
Showing results of ingestion of *Bact. abortum* suspensions

No.	Bred	Infection period began	Reacted above 1–80	Calved	Aborted	Bact. abortum			Remarks
						Milk	Fetus	Exudate	
212	Mar. 4	May 26	June 30	.....	June 25 (113)	—	+	+	Ovarian disease.
234	Mar. 6 April 11	"	June 25	.....	Sept. 25 (167)	+	+	+	Retained membranes.
238	Feb. 11 Mar. 2	"	Aug. 11	Dec. 5 (278)	.....	—	.....	—	
239	Mar. 9	"	Aug. 11	Dec. 12 (278)	.....	+	.....	+	
241	Feb. 26	"	July 14	.....	Aug. 12 (168)	+	+	+	Retained membranes.
243	Mar. 18	"	June 25	.....	Aug. 27 (162)	+	+	+	Retained membranes.
248	Feb. 18		July 7	.....	July 2 (135)	+	+	.....	
257	Mar. 8	"	Aug. 11	Dec. 4 (271)	.....	+	.....	—	Dystocia.
264	Mar. 14	"	June 25	.....	Sept. 20 (190)	—	+	+	
				CONTROLS					
214	Feb. 19 Mar. 11 April 1	.....	.....	Jan. 11 (285)	.....	—	.....	—	
215	Feb. 23 Mar. 16	.....	.....	Dec. 13 (272)	.....	—	.....	—	
218	Feb. 28	.....	.....	Nov. 28 (274)	.....	—	.....	—	
221	Feb. 18	.....	.....	Nov. 19 (275)	.....	—	.....	—	
223	Mar. 1	.....	.....	Dec. 4 (278)	.....	—	.....	—	Dystocia.
235	Feb. 23	.....	.....	Nov. 27 (280)	.....	—	.....	—	
240	Feb. 13	.....	.....	Nov. 19 (282)	.....	—	.....	—	

TABLE NO. 2
(Summary)

	Infected group	Controls
Number of animals	9	7
Became high and persistent reactors	8	0
Aborters	6	0
Bact. abortum in fetus, membranes, or both	7	0
Bact. abortum in milk	6	0
Strong vigorous calves	3	7
Genital disease following parturition	4	0
Dystocia	1	1

and demonstration of *Bact. abortum* in the uterine contents and milk in so many of the individuals are facts which incriminate the cultures fed as the cause of the infection and abortion which followed. This interpretation is further supported by the fact that cultures from the fetuses grew aerobically and quite rapidly, suggesting that their progenitors had been subjected to artificial cultivation.

We did not regularly make examination of fetal membranes and uterine exudate for organisms other than *Bact. abortum,* but all fetuses were cultured. Determinations for the presence of this organism were made by the usual method of inoculating guinea pigs. We believe the system of keeping controls is a much more reliable method of appraising the influence of other causes than is to be found in any bacteriological examination of uterine exudate or fetal membranes which can be conducted subsequent to parturition. Our controls had the same opportunity to contract genital disease that individuals in the infected group had, and they showed no manifestations suggesting serious genital infection.

We are not yet ready to say whether the difference in our results in this experiment as compared with former infection experiments in which the infectious material has been administered in capsules is explained on the basis of the method employed, or whether this difference is merely one of the inconsistencies which attend practically all infection experiments. Our belief has been that in experiments dealing with *Bact. abortum* infection of pregnant animals by ingestion, the most certain way to infect would be by means of small quantities of infectious material administered periodically for some time. In view of the rapid changes which take place within the uterus during gestation, it is probable that pregnancy is most seriously threatened when the portals of infection, as they relate to the chorion in particular, are constantly assailed. On the other hand, large single doses may at times find the chorion temporarily resistant to the infection.

Negative infection experiments, unless very carefully interpreted, may be misleading in the extreme. Positive ones carefully conducted and controlled are likely to be confirmed subsequently. If our results are verified, they furnish a method of infection of great value in the study of Bang abortion disease. This is particularly true in relation to experiments dealing with immunity and in relation to a method simulating the natural way, by which animals may be infected, later to be killed for study of specific pathologic changes which take place prior to rupture of the cervical seal.

The facts brought out by these experiments have a direct and important bearing on the practical handling of genital diseases. They show clearly that *Bact. abortum* infection takes place long after the event of conception, and brings about abortion. They remove, in some degree at least, certain doubts which have stood in the way of serious and careful efforts to segregate infected cattle, and protect sound herds.

Inspection of our herd at the time the experiment closed constituted an object lesson of great value. In the quarters of the infected group were eight heifers (one had previously been killed on account of ovarian disease) some dry, some nearly so, many still showing the effects of pre-existing metritis, only two showing a satisfactory milk yield. Three calves represented the offspring of the nine heifers for the season.

In the quarters of the controls there were seven heifers in excellent condition, each with a strong calf at her side. Six were yielding a heavy milk flow, and the other was giving a sufficient quantity to raise her calf although two quarters of her udder had failed to develop normally.

Duplication of the picture in the infected group in any breeder's herd will bring financial ruin. Conditions such as existed in the control group will enable a breeder to record a profit even in these somewhat discouraging times. *Bact. abortum* infection contracted by ingestion was responsible for the differences.

Preventive medicine makes its way but slowly. Its partial successes are recorded as failures and its complete successes sometimes command but little attention. Regardless of the proportionate part played by Bang abortion disease in the entire field of genital infection, the fact remains that the effective handling of this group of diseases must include measures based on intimate knowledge of Bang infection. This presupposes accurate diagnosis, and if preventive measures worthy of the name are to be applied they must work either in the direction of immunity, or segregation. In handling most infectious diseases the principle of segregation is our first and most reliable line of defense, and other measures apply only when this fails.

At present, members of our profession are rendering service of incalculable value in correcting genital disease which follows in the wake of Bang infection, in treating other genital infections supposedly local in character, and in the prompt elimination from our breeding herds of animals hopelessly sterile. This service must continue, or disaster awaits our cattle industry. But the more candid men agree that the best genital hygiene that can be employed will not prevent the so-called abortion storms, and our most experienced clinicians know they cannot materially stay the progress of these disasters with methods heretofore employed. Breeders are depending on the veterinary profession to develop new knowledge and more effective methods, and we in the profession may well ask ourselves if we are doing all that can be done to throw safeguards around exchange of animals among breeding herds, and in attempts to arrest outbreaks of Bang abortion disease in their beginnings. We know well the losses which attend placing infected and sound animals together. Let us learn what can be gained by consistent and painstaking efforts to keep them apart.

CONCLUSIONS

1. Infection of pregnant heifers as indicated by high and persistent agglutinin titre, abortion, and positive bacteriological findings in uterine contents and milk, was readily brought about by sprinkling the feed with *Bact. abortum* suspensions.

2. The close relation between the date when the infectious material was fed and the appearance of the manifestations just enumerated, as well as the high percentage of animals infected as compared to the completely negative record of the controls, suggests that ingestion is a common channel of infection as applied to *Bact. abortum.*

NOTES

During the course of the work there appeared certain facts, not directly related to the primary purpose of the experiments but nevertheless deserving brief mention here. Some of these will be treated more fully in a later publication.

Eleven cows exposed in past years to Bang infection, and most all thus definitely infected as indicated by agglutination tests, as well as by abortions with fetuses and uterine exudate containing *Bact. abortum,* were fed precisely the same kind of infectious material for approximately the same time as were the heifers in the infected group. Reports on these are yet incomplete, but so far no abortions have occurred among them, and most of them have calved at full term, indicating a very high resistance to Bang infection as compared to the animals in the infected group, exposed while pregnant for the first time.

During the infection period several of the heifers in the infected group developed mastitis involving one or more quarters of the udder, and *Bact. abortum* was recovered from milk samples from these quarters. In each case the inflammation disappeared after a few days or weeks. This suggests that *Bact. abortum* may at times play a part in bringing about mastitis, and that the symptom described may be an additional aid in detecting prospective aborters among heifers. No cases of this kind developed among the controls.

With one exception premonitory symptoms made it possible to predict abortions several days before they occurred.

Although fourteen of the heifers were exposed as calves to severe Bang infection, individuals in the infected group developed high and persistent agglutination reactions only after artificial infection during pregnancy, and those in the control group developed no reactions denoting infection. This is entirely in line with our observations in past years, indicating that even in herds suffering severely with Bang infection there exist in the young stock the elements of a clean herd, irrespective of exposure during fetal life or calfhood. Individual exceptions to this rule no doubt occur but so far we have not encountered them.

REFERENCES

1. HART, G. H., AND C. M. CARPENTER. Jour. A. V. M. A., Oct. 1923.
2. SCHROEDER, E. C. Jour. A. V. M. A., 1922, 13, p. 545.
3. HUDDLESON, I. F. Tech. Bul. 32, Agr. Exp. Sta., Mich. Agr. College, 1916.
4. RETTGER, L. F., G. C. WHITE AND L. M. CHAPMAN. Bul. 108, Storrs Agr. Exp. Sta., 1921, p. 37.
5. GILTNER, WARD, I. F. HUDDLESON AND R. L. TWEED. Jour. A. V. M. A. 1922, 15, p. 176.
6. SIMMS, B. T., AND F. W. MILLER. Jour. A. V. M. A., 1921, 11, p. 532.
7. CONNAWAY, J. W. Bul. 201, Agri. Exp. Sta., Univ. of Missouri, 1923, p. 4.
8. CARPENTER, C. M. Report N. Y. State Veterinary College at Cornell University, 1920–21, p. 67.
9. CARPENTER, C. M. Cornell Veterinarian, Jan., 1924.
10. WILLIAMS, W. L. Jour. A. V. M. A., May, 1924.
11. BIRCH, R. R., AND H. L. GILMAN. Report of N. Y. State Veterinary College, 1922–23.

No. 212 (*Infected Group*)

Grade Holstein heifer. Born Sept. 24, 1921. Purchased Sept. 28, 1921. Comes from small herd. No reliable history. Reared in infected herd. Removed from infected herd Nov. 2, 1922. No agglutination in any dilution until Mar. 15, 1923. (Monthly samples set.)

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1923 Feb. 15	—	—	—	—	—	—	
Mar. 15	+	—	—	—	—	—	
Apr. 16	+	+	—	—	—	—	
May 16	+	+	+	—	—	—	
June 17	+	P*	—	—	—	—	
June 16	+	+	—	—	—	—	
Aug. 15	+	P	—	—	—	—	
Sept. 15	+	P	—	—	—	—	
Oct. 16	—	—	—	—	—	—	
Nov. 15	—	—	—	—	—	—	
1924 Feb. 18	—	—	—	—	—	—	Feb. 1—Placed in experimental barn.
Mar. 15	+	—	—	—	—	—	Mar. 4—Bred to bull No. 260.
Apr. 17	+	P	—	—	—	—	
May 16	—	—	—	—	—	—	May 26—Infection period began.
June 2	—	—	—	—	—	—	
9	+	P	—	—	—	—	
16	+	—	—	—	—	—	
23	+	+	—	—	—	—	June 25—Cow aborted. Membranes not retained. Fetus and uterine exudate contained *Bact. abortum.* Milk negative.
30	+	+	+	+	+	—	
July 7	+	+	+	+	+	—	
14	+	+	+	+	+	P	
21	+	+	+	+	—	—	
28	+	+	+	+	+	+	July 24—Bred to Bull No. 261. Did not conceive.
Aug. 11	+	+	+	+	+	+	
18	+	+	+	+	+	+	
25	+	+	+	+	+	+	Aug. 27—Infection period ended.
Sept. 1	+	+	+	+	+	+	
8	+	+	+	+	+	+	
15	+	+	+	+	+	+	
22	+	+	+	+	+	+	
29	+	+	+	+	+	+	
Oct. 6	+	+	+	+	+	+	
13	+	+	+	+	+	+	
20	+	+	+	+	+	+	
27	+	+	+	+	+	+	
Nov. 3	+	+	+	+	+	+	Nov. 18—Killed.

* P = Partial.

No. 214 (*Control*)

Grade Guernsey. Born Oct. 19, 1921. Dam a non-reactor. Heifer has never been in an infected herd. Agglutinations to Mar. 15, 1923, negative in all dilutions.

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1923 Feb. 15							
Mar. 15	—	—	—	—	—	—	
Apr. 16	+	—	—	—	—	—	
May 16	—	—	—	—	—	—	
June 17	+	—	—	—	—	—	
July 16	—	—	—	—	—	—	
Aug. 15	+	+	—	—	—	—	
Sept. 15	+	+	—	—	—	—	
Oct. 16	—	—	—	—	—	—	
Nov. 15	—	—	—	—	—	—	Feb. 1—Placed in experimental barn.
1924 Feb. 18	—	—	—	—	—	—	Feb. 19—Bred to bull No. 260.
Mar. 15	—	—	—	—	—	—	Mar. 11—Bred to bull No. 260.
Apr. 17	—	—	—	—	—	—	Apr. 1—Bred to bull No. 260.
May 16	—	—	—	—	—	—	May 26—Control in infection ex-
June 2	—	—	—	—	—	—	periment.
9	—	—	—	—	—	—	
16	—	—	—	—	—	—	
23	—	—	—	—	—	—	
30	—	—	—	—	—	—	
July 7	—	—	—	—	—	—	
14	—	—	—	—	—	—	
21	+	—	—	—	—	—	
28	—	—	—	—	—	—	
Aug. 11	P	—	—	—	—	—	
18	—	—	—	—	—	—	
25	P	P	—	—	—	—	
Sept. 1	—	—	—	—	—	—	
8	—	—	—	—	—	—	
15	P	—	—	—	—	—	
22	—	—	—	—	—	—	
29	P	P	—	—	—	—	
Oct. 6	+	P	—	—	—	—	
13	+	—	—	—	—	—	
20	+	—	—	—	—	—	
27	+	P	—	—	—	—	
Nov. 3	+	—	—	—	—	—	
10	+	+	+	—	—	—	
17	P	—	—	—	—	—	Jan. 11—Calved (285). Normal
24	+	—	—	—	—	—	parturition. *Bact. abortum* not in
Dec. 1	+	—	—	—	—	—	milk or fetal membranes.

No. 215 (*Control*)

Grade Holstein. Purchased Nov. 17, 1921, three days after birth, and placed in Bang infected herd. Monthly agglutinations in 1922 negative.

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1923 Feb. 15	—	—	—	—	—	—	
Mar. 15	—	—	—	—	—	—	
Apr. 16	+	+	—	—	—	—	
May 16	—	—	—	—	—	—	
June 17	+	—	—	—	—	—	
July 16	+	—	—	—	—	—	
Aug. 15	+	P	—	—	—	—	
Sept. 15	+	+	P	—	—	—	
Oct. 16	+	+	—	—	—	—	Nov. 2—Removed from infected herd.
Nov. 15	+	—	—	—	—	—	Feb. 1—Placed in experimental barn.
1924 Feb. 18	+	—	—	—	—	—	Feb. 23—Bred to bull No. 260.
Mar. 15	—	—	—	—	—	—	Mar. 16—Bred to bull No. 260.
Apr. 17	+	—	—	—	—	—	
May 16	+	+	—	—	—	—	May 26—Control in infection experiment.
June 2	—	—	—	—	—	—	
9	—	—	—	—	—	—	
16	—	—	—	—	—	—	
23	—	—	—	—	—	—	
30	P	P	—	—	—	—	
July 7	+	—	—	—	—	—	
14	+	—	—	—	—	—	
21	—	—	—	—	—	—	
28	—	—	—	—	—	—	
Aug. 11	P	P	—	—	—	—	
18	—	—	—	—	—	—	
25	P	P	—	—	—	—	
Sept. 1	—	—	—	—	—	—	
8	—	—	—	—	—	—	
15	—	—	—	—	—	—	
22	—	—	—	—	—	—	
29	P	P	—	—	—	—	
Oct. 6	—	—	—	—	—	—	
13	—	—	—	—	—	—	
20	P	—	—	—	—	—	
27	P	P	—	—	—	—	
Nov. 3	—	—	—	—	—	—	Dec. 13—Calved (272). Placenta not retained. Milk flow good. Calf strong. Milk and fetal membranes do not contain *Bact. abortum*.
10	+	—	—	—	—	—	
17	—	—	—	—	—	—	
24	—	—	—	—	—	—	
Dec. 1	—	—	—	—	—	—	

No. 218 (*Control*)

Grade Guernsey. Born Dec. 21, 1921. Dam a reactor. *Bact. abortum* in uterine exudate. Calf reared in infected herd. Monthly agglutinations in 1922 negative. Removed from infected herd on Nov. 2, 1922.

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1923 Feb. 15	—	—	—	—	—	—	
Mar. 15	—	—	—	—	—	—	
Apr. 16	+	—	—	—	—	—	
May 16	—	—	—	—	—	—	
June 17	+	—	—	—	—	—	
July 16	+	—	—	—	—	—	
Aug. 15	+	P	—	—	—	—	
Sept. 15	+	+	—	—	—	—	
Oct. 16	+	—	—	—	—	—	
Nov. 15	—	—	—	—	—	—	Feb. 1—Placed in experimental barn.
1924 Feb. 18	+	+	—	—	—	—	Feb. 28—Bred to bull No. 260.
Mar. 15	—	—	—	—	—	—	
Apr. 17	—	—	—	—	—	—	
May 16	—	—	—	—	—	—	May 26—Control in infection ex-
June 2	—	—	—	—	—	—	periment.
9	—	—	—	—	—	—	
16	—	—	—	—	—	—	
23	—	—	—	—	—	—	
30	—	—	—	—	—	—	
July 7	—	—	—	—	—	—	
14	—	—	—	—	—	—	
21	—	—	—	—	—	—	
28	—	—	—	—	—	—	
Aug. 11	—	—	—	—	—	—	
18	—	—	—	—	—	—	
25	—	—	—	—	—	—	
Sept. 1	+	—	—	—	—	—	
8	—	—	—	—	—	—	
15	—	—	—	—	—	—	
22	P	—	—	—	—	—	
29	+	—	—	—	—	—	
Oct. 6	+	—	—	—	—	—	
13	—	—	—	—	—	—	
20	—	—	—	—	—	—	
27	—	—	—	—	—	—	
Nov. 3	+	—	—	—	—	—	
10	—	—	—	—	—	—	
17	+	P	—	—	—	—	Nov. 28—Calved normally (274).
24	—	—	—	—	—	—	Membranes and milk negative to
Dec. 1	—	—	—	—	—	—	*Bact. abortum*.

No. 221 (*Control*)

Grade Holstein. Born Feb. 22, 1922, in experimental barn. No examination of dam's uterine exudate for *Bact. abortum*. Monthly agglutination of calf's blood in 1922 negative.

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1923 Feb. 15	—	—	—	—	—	—	
Mar. 15	+	P	—	—	—	—	
Apr. 16	—	—	—	—	—	—	
May 16	+	+	+	—	—	—	
June 17	—	—	—	—	—	—	
July 16	+	—	—	—	—	—	
Aug. 15	—	—	—	—	—	—	
Sept. 15	—	—	—	—	—	—	
Oct. 16	+	+	+	—	—	—	
Nov. 15	—	—	—	—	—	—	
1924 Feb. 18	+	—	—	—	—	—	Feb. 18—Bred to bull No. 260.
Mar. 15	—	—	—	—	—	—	
Apr. 17	—	—	—	—	—	—	
May 16	—	—	—	—	—	—	May 26—Control in infection ex-
June 2	—	—	—	—	—	—	periment.
9	—	—	—	—	—	—	
16	—	—	—	—	—	—	
23	—	—	—	—	—	—	
30	P	—	—	—	—	—	
July 7	P	—	—	—	—	—	
14	—	—	—	—	—	—	
21	P	P	—	—	—	—	
28	—	—	—	—	—	—	
Aug. 11	—	—	—	—	—	—	
18	P	—	—	—	—	—	
25	P	P	—	—	—	—	
Sept. 1	—	—	—	—	—	—	
8	—	—	—	—	—	—	
15	—	—	—	—	—	—	
22	—	—	—	—	—	—	
29	—	—	—	—	—	—	
Oct. 6	P	P	—	—	—	—	
13	—	—	—	—	—	—	
20	—	—	—	—	—	—	
27	—	—	—	—	—	—	
Nov. 3	—	—	—	—	—	—	
10	—	—	—	—	—	—	Nov. 19—Calved (275). Calf large
24	—	—	—	—	—	—	and vigorous. Normal parturition. Membranes and milk negative to
Dec. 1	—	—	—	—	—	—	*Bact. abortum*.

No. 223 (*Control*)

Grade Holstein. Born May 15, 1922. *Bact. abortum* not in the uterine exudate of dam at time of parturition. Calf reared in infected herd. Removed from infected herd Nov. 2, 1922. Monthly agglutinations in 1922 negative.

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1923 Feb. 15							
Mar. 15	+	—	—	—	—	—	
Apr. 16	+	+	+	—	—	—	
May 16	—	—	—	—	—	—	
June 17	—	—	—	—	—	—	
July 16	—	—	—	—	—	—	
Aug. 15	+	—	—	—	—	—	
Sept. 15	+	+	—	—	—	—	
Oct. 16							
Nov. 15	—	—	—	—	—	—	
1924 Feb. 18	+	—	—	—	—	—	Feb. 1—Placed in experimental barn.
Mar. 15	+	+	+	—	—	—	Mar. 1—Bred to bull No. 260.
Apr. 17	—	—	—	—	—	—	
May 16	—	—	—	—	—	—	May 26—Control in infection ex-
June 2	—	—	—	—	—	—	periment.
9	—	—	—	—	—	—	
16	—	—	—	—	—	—	
23	—	—	—	—	—	—	
30	P	—	—	—	—	—	
July 7	—	—	—	—	—	—	
14	—	—	—	—	—	—	
21	—	—	—	—	—	—	
28	—	—	—	—	—	—	
Aug. 11	—	—	—	—	—	—	
18	—	—	—	—	—	—	
25	—	—	—	—	—	—	
Sept. 1	—	—	—	—	—	—	
8	—	—	—	—	—	—	
15	—	—	—	—	—	—	
22	—	—	—	—	—	—	
29	P	—	—	—	—	—	
Oct. 6	—	—	—	—	—	—	
13	—	—	—	—	—	—	
20	—	—	—	—	—	—	
27	—	—	—	—	—	—	
Nov. 3	—	—	—	—	—	—	
10	+	P	—	—	—	—	Dec. 4 — Calved (278).. Dystocia.
17	—	—	—	—	—	—	Membranes not retained. Mem-
24	—	—	—	—	—	—	branes and milk negative to *Bact.*
Dec. 1							*abortum*.

No. 234 (*Infected Group*)

Grade Guernsey. Born June 29, 1922. Dam a reactor. *Bact. abortum* not in uterine contents when calf was born.

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1922 July 20	+	+	+	+	+		
Aug. 19				—	—		
Sept. 18							
Oct. 20	—	—	—	—	—	—	
Nov. 24	—	—	—	—	—		
1923 Jan. 19	—	—	—	—	—		
Feb. 15	—	—	—	—	—	—	
Mar. 15	+	—	—	—	—	—	
Apr. 16	+	+	—	—	—	—	
May 16	+	—	—	—	—	—	
June 17	+	—	—	—	—	—	
July 16	+	—	—	—	—	—	
Aug. 15	+	—	—	—	—	—	
Sept. 15	+	+	—	—	—	—	
Oct. 16	—	—	—	—	—	—	
Nov. 15	—	—	—	—	—	—	Nov. 2—Removed from infected herd.
1924 Feb. 18	+	—	—	—	—	—	Feb. 1—Placed in experimental barn.
Mar. 15	—	—	—	—	—	—	Mar. 6—Bred to bull No. 260.
Apr. 17	+	—	—	—	—	—	Apr. 11—Bred to bull No. 260.
May 16	—	—	—	—	—	—	May 26—Infection period began.
June 9	P	P	—	—	—	—	
16	+	+	+	—	—	—	
25	+	+	+	+	+	+	
30	+	+	+	+	+	+	
July 7	+	+	+	+	+	+	
14	+	+	+	+	—	—	
21	+	+	+	+	—	—	
28	+	+	+	—	—	—	
Aug. 11	+	+	+	+	—	—	
18	+	+	+	P	—	—	
25	+	+	+	+	—	—	Aug. 27—Infection period ended.
Sept. 1	+	+	+	+	—	—	
8	+	+	+	+	+	+	
15	+	+	+	+	+	+	
22	+	+	+	+	+	+	
29	+	+	+	+	+	+	Sept. 25—Aborted. Retained membranes. Fetus, exudate, and milk contained *Bact. abortum*.
Oct. 6	+	+	+	+	+	+	
13	+	+	+	+	P	—	
20	+	+	+	+	—	—	
27	+	+	+	+	+	—	
Nov. 3	+	+	+	+	+	P	

No. 235 (*Control*)

Grade Holstein. Born July 6, 1922. Dam a reactor. *Bact. abortum* not in uterine exudate. Calf's blood agglutinated above 1–100 for three consecutive months, then was negative in dilutions as low as 1–20 until Mar. 15, 1923. Removed from infected herd on Nov. 2, 1922.

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1923 Feb. 15	—	—	—	—	—	—	
Mar. 15	—	—	—	—	—	—	
Apr. 16	+	P	—	—	—	—	
May 16	+	P	—	—	—	—	
June 17	+	—	—	—	—	—	
July 16	+	P	—	—	—	—	
Aug. 15	+	P	—	—	—	—	
Sept. 15	+	+	—	—	—	—	
Oct. 16	—	—	—	—	—	—	
Nov. 15	+	—	—	—	—	—	Feb. 1—Placed in experimental herd.
1924 Feb. 18	+	+	—	—	—	—	Feb. 23—Bred to bull No. 260.
Mar. 15	+	+	+	+*	—	—	
Apr. 17	—	—	—	—	—	—	
May 16	—	—	—	—	—	—	May 26—Control in infection ex-
June 2	—	—	—	—	—	—	periment.
9	—	—	—	—	—	—	
16	—	—	—	—	—	—	
23	—	—	—	—	—	—	
30	—	—	—	—	—	—	
July 7	—	—	—	—	—	—	
14	—	—	—	—	—	—	
21	—	—	—	—	—	—	
28	—	—	—	—	—	—	
Aug. 11	—	—	—	—	—	—	
18	—	—	—	—	—	—	
25	—	—	—	—	—	—	
Sept. 1	+	P	—	—	—	—	
8	—	—	—	—	—	—	
15	+	—	—	—	—	—	
22	—	—	—	—	—	—	
29	—	—	—	—	—	—	
Oct. 6	—	—	—	—	—	—	
13	—	—	—	—	—	—	
20	—	—	—	—	—	—	
27	—	—	—	—	—	—	
Nov. 3	—	—	—	—	—	—	Nov. 27 — Calved (280). Large,
10	+	+	—	—	—	—	vigorous calf. Normal parturition.
17	—	—	—	—	—	—	Membranes and milk negative to
24	—	—	—	—	—	—	*Bact. abortum*.
Dec. 1	—	—	—	—	—	—	

* Probable clerical error in recording.

No. 238 (*Infected Group*)

Grade Guernsey. Born Dec. 2, 1922. Dam a reactor. Uterine exudate did not contain *Bact. abortum* following birth of calf.

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1923 Mar. 15	+	+	—	—	—	—	
Apr. 16	+	—	—	—	—	—	
May 16	+	—	—	—	—	—	
June 17	+	—	—	—	—	—	
July 16	+	—	—	—	—	—	
Aug. 15	+	+	—	—	—	—	
Sept. 15	+	+	+	—	—	—	
Oct. 16	—	—	—	—	—	—	
Nov. 15	—	—	—	—	—	—	Nov. 2—Removed from infected herd.
1924 Feb. 18	—	—	—	—	—	—	Feb. 1—Placed in experimental barn.
Mar. 15	—	—	—	—	—	—	Feb. 11—Bred to bull No. 260.
Apr. 17	—	—	—	—	—	—	Mar. 2—Bred to bull No. 260.
May 16	—	—	—	—	—	—	May 26—Infection period began.
June 2	—	—	—	—	—	—	
9	—	—	—	—	—	—	
16	—	—	—	—	—	—	
23	+	+	—	—	—	—	June 27—Marked swelling of two
30	+	+	P	—	—	—	left quarters of udder.
July 7	+	+	P	—	—	—	
14	+	+	+	—	—	—	
21	+	+	P	—	—	—	
28	+	+	+	—	—	—	
Aug. 11	+	+	+	+	—	—	
18	+	+	P	—	—	—	
25	+	+	+	—	—	—	Aug. 27—Infection period ended.
Sept. 1	+	+	—	—	—	—	
8	+	+	—	—	—	—	
15	+	+	—	—	—	—	
22	+	+	—	—	—	—	
29	+	+	+	—	—	—	
Oct. 6	+	+	—	—	—	—	
13	—	—	—	—	—	—	
20	—	—	—	—	—	—	
27	—	—	—	—	—	—	
Nov. 3	—	—	—	—	—	—	
10	+	—	—	—	—	—	
17	+	P	—	—	—	—	Dec. 5—Cow calved (278). Milk and
24	—	—	—	—	—	—	membranes negative to *Bact. abortum.*
Dec. 1	—	—	—	—	—	—	

No. 239 (*Infected Group*)

Grade Holstein. Born Jan. 10, 1923. Dam not a reactor. *Bact. abortum* not in uterine exudate at time of parturition.

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1923 Mar. 15	—	—	—	—	—	—	
Apr. 16	—	—	—	—	—	—	
May 16	+	+	—	—	—	—	
June 17	—	—	—	—	—	—	
July 16	+	—	—	—	—	—	
Aug. 15	—	—	—	—	—	—	
Sept. 15	—	—	—	—	—	—	
Oct. 16	—	—	—	—	—	—	
Nov. 15	—	—	—	—	—	—	Nov. 2—Removed from infected herd.
1924 Feb. 18	+	—	—	—	—	—	Feb. 1—Placed in experimental barn.
Mar. 15	—	—	—	—	—	—	Mar. 9—Bred to bull No. 260.
Apr. 17	—	—	—	—	—	—	
May 16	+	—	—	—	—	—	May 26—Infection period began.
June 2	—	—	—	—	—	—	
9	—	—	—	—	—	—	
16	—	—	—	—	—	—	
25	+	+	—	—	—	—	
30	+	+	P	—	—	—	
July 7	+	+	+	—	—	—	July 16—Marked swelling in left hind
14	+	+	+	—	—	—	quarter of udder.
21	+	+	+	—	—	—	
28	+	+	+	—	—	—	July 28—Milk sample positive to
Aug. 11	+	+	+	+	P	P	*Bact. abortum*. (Left hind quarter.)
18	+	+	+	+	+	—	
25	+	+	+	+	—	—	Aug. 27—Infection period ended.
Sept. 1	+	+	+	+	—	—	
8	+	+	+	+	+	+	
15	+	+	+	+	+	+	
22	+	+	+	+	+	+	
29	+	+	+	+	+	+	
Oct. 6	+	+	+	+	—	—	
13	+	+	+	P	—	—	
20	+	+	+	+	+	—	
27	+	+	+	+	—	—	
Nov. 3	+	+	+	+	+	—	
10	+	+	+	+	+	—	
17	+	+	+	+	—	—	Dec. 12—Cow calved (278). *Bact.*
24	+	+	+	—	—	—	*abortum* in milk and fetal mem-
Dec. 1	+	+	P	—	—	—	branes.

No. 240 (*Control*)

Grade Holstein. Born Feb. 16, 1923, in experimental barn. Dam a reactor. Uterine exudate contained *Bact. abortum.*

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1923 Feb. 15							
Mar. 15	+	+	+	—	—	—	
Apr. 16	+	+	—	—	—	—	
May 16	+	+	P	—	—	—	
June 17	+	—	—	—	—	—	
July 16	+	P	—	—	—	—	
Aug. 15	+	—	—	—	—	—	
Sept. 15	—	—	—	—	—	—	
Oct. 16	—	—	—	—	—	—	
Nov. 15	—	—	—	—	—	—	
1924 Feb. 18	—	—	—	—	—	—	Feb. 13—Bred to bull No. 260.
Mar. 15	—	—	—	—	—	—	
Apr. 17	—	—	—	—	—	—	
May 16	—	—	—	—	—	—	May 26—Control in infection experiment.
June 2	—	—	—	—	—	—	
9	+	P	—	—	—	—	
16	+	P	—	—	—	—	
23	—	—	—	—	—	—	
30	P	—	—	—	—	—	
July 7	—	—	—	—	—	—	
14	—	—	—	—	—	—	
21	—	—	—	—	—	—	
28	+	—	—	—	—	—	
Aug. 11	—	—	—	—	—	—	
18	—	—	—	—	—	—	
25	+	P	—	—	—	—	
Sept. 1	—	—	—	—	—	—	
8	—	—	—	—	—	—	
15	—	—	—	—	—	—	
22	P	P	—	—	—	—	
29	P	P	—	—	—	—	
Oct. 6	+	—	—	—	—	—	
13	—	—	—	—	—	—	
20	—	—	—	—	—	—	
27	—	—	—	—	—	—	
Nov. 3	—	—	—	—	—	—	
10	+	—	—	—	—	—	Nov. 19 — Calved (282). Placenta not retained. Milk flow comparatively light. Membranes and milk negative to *Bact. abortum.*
17	—	—	—	—	—	—	
24	+	—	—	—	—	—	
Dec. 1	—	—	—	—	—	—	

No. 241 (*Infected Group*)

Grade Holstein. Born Feb. 25, 1923. Reacting dam. *Bact. abortum* was not in uterine contents at time calf was born.

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1923 Mar. 15	+	+	+	+	+	+	Calf sucking No. 188, a reactor. In
Apr. 16	+	+	+	—	—	—	infected herd.
May 16	—	—	—	—	—	—	
June 17	+	P	—	—	—	—	
July 16	+	—	—	—	—	—	
Aug. 15	+	—	—	—	—	—	
Sept. 15	+	+	—	—	—	—	
Oct. 16	+	—	—	—	—	—	
Nov. 15	—	—	—	—	—	—	Nov. 2—Removed from infected herd.
1924 Feb. 18	—	—	—	—	—	—	Feb. 1—Placed in experimental barn.
Mar. 15	—	—	—	—	—	—	Feb. 26—Bred to bull No. 260.
Apr. 17	—	—	—	—	—	—	
May 16	—	—	—	—	—	—	May 26—Infection period began.
June 2	—	—	—	—	—	—	
9	—	—	—	—	—	—	
16	+	+	—	—	—	—	
23	+	+	P	—	—	—	
30	+	+	+	—	—	—	
July 7	+	+	P	—	—	—	
14	+	+	+	+	+	+	July 13—Marked swelling of two
21	+	+	+	+	+	+	rear quarters of udder.
28	+	+	+	+	+	+	
Aug. 11	+	+	+	+	+	+	Aug. 12—Aborted. Retained mem-
18	+	+	+	+	+	+	branes. Milk, fetus, and membranes
18	+	+	+	+	+	+	contained *Bact. abortum*.
25	+	+	+	+	+	+	
Sept. 1	+	+	+	+	+	+	
8	+	+	+	+	+	+	Aug. 27—Infection period ended.
15	+	+	+	+	+	+	
22	+	+	+	+	+	+	
29	+	+	+	+	+	+	
Oct. 6	+	+	+	+	+	+	
13	+	+	+	+	+	+	
20	+	+	+	+	+	+	
27	+	+	+	+	+	+	
Nov. 3	+	+	+	+	+	+	
10	+	+	+	+	+	+	
17	+	+	+	+	+	+	
24	+	+	+	—	—	—	
Dec. 1	+	+	+	+	P	—	

No. 243 (*Infected Group*)

Grade Holstein. Born Mar. 3, 1923, in experimental barn. *Bact. abortum* in uterine exudate of dam. Dam a high reactor.

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1923 Mar. 15	+	—	—	—	—	—	
June 17	+	+	—	—	—	—	
July 16	+	+	—	—	—	—	
Aug. 15	+	P	—	—	—	—	
Sept. 15	+	+	—	—	—	—	
Oct. 16	+	—	—	—	—	—	
1924 Feb. 18	+	—	—	—	—	—	
Mar. 15	—	—	—	—	—	—	Mar. 18—Bred to bull No. 260.
Apr. 17	—	—	—	—	—	—	
May 16	—	—	—	—	—	—	May 26—Infection period began.
June 2	—	—	—	—	—	—	
9	+	+	—	—	—	—	
16	+	+	P	—	—	—	
25	+	+	+	+	—	—	
30	+	+	+	+	+	+	
July 7	+	+	+	+	+	—	
14	+	+	+	—	—	—	
21	+	+	+	+	+	+	
28	+	+	+	+	+	P	July 28—Milk sample from left hind quarter positive to *Bact. abortum.* (Quarter swollen.)
Aug. 11	+	+	+	+	+	+	
18	+	+	+	+	+	+	
25	+	+	+	+	+	+	Aug. 27—Infection period ended. Cow aborted. Retained placenta. Fetus, milk, and placenta contained *Bact. abortum.*
Sept. 1	+	+	+	+	+	+	
8	+	+	+	+	+	+	
15	+	+	+	+	+	+	
22	+	+	+	+	+	+	
29	+	+	+	+	+	+	
Oct. 6	+	+	+	+	+	+	
13	+	+	+	+	+	+	
20	+	+	+	+	+	+	
27	+	+	+	+	+	+	
Nov. 3	+	+	+	+	+	+	
10	+	+	+	+	+	+	
17	+	+	+	+	—	—	
Dec. 1	+	+	+	+	P	—	

No. 248 (*Infected Group*)

Born April 13, 1923, in experimental barn. Dam a non-reactor. Uterine contents negative to *Bact. abortum* at time calf was born.

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1923 Apr. 16	—	—	—	—	—	—	
May 16	—	—	—	—	—	—	
June 17	—	—	—	—	—	—	
July 16	—	—	—	—	—	—	
Aug. 15	—	—	—	—	—	—	
Sept. 15	—	—	—	—	—	—	
Oct. 16	—	—	—	—	—	—	
Nov. 15	—	—	—	—	—	—	
1924 Feb. 18	—	—	—	—	—	—	Feb. 18—Bred to bull No. 260.
Mar. 15	—	—	—	—	—	—	
Apr. 17	—	—	—	—	—	—	
May 16	—	—	—	—	—	—	May 26—Infection period began.
June 2	—	—	—	—	—	—	
9	—	—	—	—	—	—	
16	—	—	—	—	—	—	June 25—Right hind quarter of udder
23	+	+	+	P	—	—	showed marked swelling.
30	+	+	+	P	—	—	
July 7	+	+	+	+	+	P	July 2—Aborted. Membranes not
14	+	+	+	+	+	+	retained. Fetus and milk contained
21	+	+	+	+	+	+	*Bact. abortum*.
28	+	+	+	+	+	+	
Aug. 11	+	+	+	+	+	+	
18	+	+	+	+	+	+	
25	+	+	+	+	+	+	Aug. 27—Infection period ended.
Sept. 1	+	+	+	+	+	+	
8	+	+	+	+	+	+	
15	+	+	+	+	+	+	
22	+	+	+	+	+	+	
29	+	+	+	+	+	+	
Oct. 6	+	+	+	+	+	+	
13	+	+	+	+	+	+	

No. 257 (*Infected Group*)

Grade Holstein. Born about Dec. 20, 1922. Had been used in attempt to produce calf pneumonia artificially. Calf had suffered from the effects of the inoculation, but was thrifty at the time experiment began. Began to run down in condition when placed with the other animals.

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1923 Aug. 15	—	—	—	—	—	—	
Sept. 15	P	—	—	—	—	—	
Oct. 16	—	—	—	—	—	—	
Nov. 15	—	—	—	—	—	—	
Feb. 18	—	—	—	—	—	—	
Mar. 15	—	—	—	—	—	—	Mar. 8—Bred to bull No. 260.
Apr. 17	—	—	—	—	—	—	
May 16	—	—	—	—	—	—	May 26—Infection period began.
June 2	—	—	—	—	—	—	
9	—	—	—	—	—	—	
16	—	—	—	—	—	—	
25	+	+	—	—	—	—	
30	P	P	—	—	—	—	
July 7	+	+	—	—	—	—	
14	+	+	—	—	—	—	
21	+	—	—	—	—	—	
28	+	+	+	—	—	—	
Aug. 11	+	+	+	+	+	+	
18	+	+	+	+	+	+	
25	+	+	+	+	+	+	Aug. 27—Infection period ended.
Sept. 1	+	+	+	+	+	+	
15	+	+	+	+	+	+	
22	+	+	+	+	+	+	
29	+	+	+	+	+	+	
Oct. 6	+	+	+	+	—	—	
13	+	+	P	—	—	—	
20	+	+	—	—	—	—	
27	+	+	+	+	—	—	
Nov. 3	+	+	+	P	—	—	
10	+	+	+	+	—	—	
17	+	+	+	+	—	—	Dec. 4—Cow calved (271). Dystocia. Membranes negative and milk positive to *Bact. abortum.*
24	+	+	+	—	—	—	
Dec. 1	+	+	+	—	—	—	

No. 264 (*Infected Group*)

Grade Holstein. Born August 15, 1922. Purchased Nov. 2, 1924, and moved about ¼ mile into experimental barn. Blood had failed to agglutinate *Bact. abortum* antigen a short time prior to purchase.

AGGLUTINATION — BACTERIUM ABORTUM ANTIGEN

Date Drawn	Dilution						Remarks
	20	40	80	160	320	640	
1924 Feb. 18	—	—	—	—	—	—	
Mar. 15	—	—	—	—	—	—	Mar. 14—Bred to bull No. 260.
Apr. 17	—	—	—	—	—	—	
May 16	—	—	—	—	—	—	May 26—Infection period began.
June 2	—	—	—	—	—	—	
9	—	—	—	—	—	—	
16	—	—	—	—	—	—	
25	+	+	+	+	+	—	
30	+	+	+	+	+	+	
July 7	+	+	+	+	+	+	July 3—Marked swelling of right hind
14	+	+	+	+	+	—	quarter of udder.
21	+	+	+	+	—	—	
28	+	+	+	+	—	—	
Aug. 11	+	+	+	—	—	—	
14	+	+	+	—	—	—	
18	+	+	+	—	—	—	
25	+	+	+	—	—	—	Aug. 27—Infection period ended.
Sept. 1	+	+	+	—	—	—	
8	+	+	+	+	—	—	
15	+	+	+	+	—	—	
22	+	+	+	+	+	P	Sept. 20—Aborted. Membranes not
29	+	+	+	+	+	+	retained. Uterine exudate and fetus
Oct. 6	+	+	+	+	+	+	contained *Bact. abortum*. Milk nega-
13	+	+	+	+	+	+	tive.
20	+	+	+	+	+	+	
27	+	+	+	+	+	+	
Nov. 3	+	+	+	+	+	+	
10	+	+	+	+	+	+	
17	+	+	+	+	+	+	
24	+	+	+	+	+	+	

A MOULD ASSOCIATED WITH ABORTION IN CATTLE*

By H. L. GILMAN and R. R. BIRCH
Veterinary Experiment Station

In 1920 Dr. Theobald Smith[1] reported finding a mould of the genus Mucor associated with disease of the fetal membranes. He made the discovery by accident while culturing the pregnant uteri of several cows slaughtered at an abattoir. The tract from which the mould was recovered was apparently normal externally, and the uterine seal intact, thus eliminating the probability of contamination. Pure cultures were obtained from the chorion and amniotic fluid, and from the lungs and digestive tract of the fetus. The cotyledons revealed a more or less characteristic type of necrosis involving the stem and point of attachment to the uterine wall. Guinea-pigs inoculated with the uterochorionic fluid, amniotic fluid, and the abomasum contents of the fetus failed to demonstrate the presence of *Bact. abortum.* While the animal had not actually aborted, Smith stated: "The condition of the amniotic fluid and the contents of the rumen of the fetus justify the inference that premature expulsion was impending."

This work to us was very interesting, for even though infection with the organism does not seem to be very widespread, any factor having a relation to the complex problem of abortion and disease of the genital organs helps to give us a clearer understanding of the conditions that have to be contended with. The question of etiology is always of fundamental importance, and in the study of this problem it seems we have laid too little stress on the study of the bacteriology of the fetus and pregnant uterus before the breaking down of the uterine seal. As stated by Smith: "An accurate knowledge of the number and variety of living organisms which may invade the utero-chorionic space during pregnancy in bovines and multiply enough to set up localized or general disease of the chorion can only be gained by a cumulative study of the pregnant uterus before discharge of the fetus. After discharge of the latter, adhesion of the placenta, the loss of pathological fluids held between the uterine wall and chorion, the soiling of the placenta by the bedding, or its destruction by the cow, all stand in the way of an elucidation of those still unknown agencies of disease which may be of far more significance than is credited to them."

Later developments show that perhaps the moulds play a somewhat more important role in the etiology of abortion than is generally believed. As a rule they are non-pathogenic, and, as far as we know, the above-mentioned case is the only one in which this organism has been found associated with abortion.

Early in 1921 Carpenter[2] recovered a mould in pure culture

* The Cornell Veterinarian, Vol. XV (1925), p. 81.
[1] Jour. Exper. Medicine, February 1, 1920, xxxi, No. 2, 115–122.
[2] Report N. Y. S. Veterinary College, 1920–21.

from the digestive tract of a fetus brought in for diagnosis. No notations were made as to the characteristics of the mould other than that it formed a heavy basement membrane adhering closely to the medium, covered by wavy interlacing aerial filaments of a bluish tinge. The fetus was a 7 months' abort in good condition. The stomach contents were described as a cloudy viscid fluid containing pellets of meconium, small masses of epithelial cells, and yellowish pus-like flocculi.

In December 1922, a fetus was brought in for routine diagnosis. The fetus represented a six months' abortion, with prolapse of the uterus. A mould was recovered in pure culture from the lungs, and all parts of the digestive tract of the fetus. The thoracic and abdominal cavities were filled with a thin reddish fluid. The abomasum contained a thick opalescent mucus with some cream-colored clumps. Blood from the dam failed to reveal the presence of agglutinins for *Bact. abortum.*

Almost one year later, in November, 1923, another fetus was brought in. Abortion occurred at six and a half months, with retention of the membranes. The fetus was small and poorly nourished. A mould was also recovered from the lungs and digestive tract of this fetus. The abomasum contained a slightly cloudy straw-colored mucus. The dam's blood was likewise negative to the agglutination test for infection with *Bact. abortum.*

Guinea-pig inoculations from all three fetuses failed to show the presence of *Bact. abortum.* All three strains of the organism were identical as far as could be determined. The placentas were not brought in, so their pathology could not be noted.

The three fetuses were from the same herd, one which is under the supervision of the Department of Medicine. The herd consists of about 90 animals, and throughout the period covered by these abortions the entire herd gave negative reactions to the agglutination test for *Bact. abortum.* Abortions averaged about 10 per cent yearly. Other diseases of the genital organs were of moderate intensity.

The possibility of infection of the fetus with the organism after expulsion could not be absolutely ruled out. However, the three abortions in question occurred in cold weather, and the fetuses were brought to the laboratory within a few hours. Also, but one other mould has been recovered from other fetuses during or since this period. In this one instance it appeared in conjunction with *B. coli.* While the organism might not have been the direct cause of the abortions, it was without doubt intimately associated with them, particularly in view of the fact that *Bact. abortum* was ruled out both by cultures and guinea-pig inoculations.

Feeling fairly well convinced that the mould was very closely associated with the three abortions, we determined to attempt producing the disease under experimental conditions. It was decided to inject animals intravenously when they were fairly well along in pregnancy. While the intravenous method of infection is by no means the most satisfactory, we thought it best suited to our

preliminary tests. Accordingly, on January 8, 1924, we injected cow No. 88 with 6 c. c. of a fairly heavy suspension of the mould. The suspension was prepared by grinding up agar growths in a mortar with sterile sand, adding physiological saline solution, and filtering through cotton. The animal selected was 12 years old and had an excellent breeding record. Until 1923 she had not aborted nor shown evidence of any genital infection, nor had she reacted to the agglutination tests for *Bact. abortum*. In 1923 she was injected with a suspension of *Bact. abortum*, aborted, and gave a high agglutinin titre. At the time of the injection, January 8, 1924, the blood did not agglutinate in any dilution. The history, subsequent to inoculation with the mould, follows:

Jan. 9. Cow somewhat uneasy and recumbent most of the time. Respiration increased. Temperature 101.3° F.

Jan. 10. Cow down most of the morning. There was a pronounced weakness in the hind legs, suggesting a thrombus. Respiration and pulse were somewhat accelerated. Temperature 102° F. Appetite was good and rumination active.

Jan. 11. Condition about the same. The cow was raised to her feet with the aid of slings. Appetite was fair and rumination active. Temperature 101.8° F.

Jan. 13. Marked evidence of improvement. The weakness in hind legs still continued. Appetite was good, and the animal showed no distress. Rumination active.

Jan. 18. The knees were somewhat swollen due apparently to contusions resulting from attempts to rise. She was helped to her feet. Appetite and digestion were normal.

Jan. 22. The swelling had extended from the knees to the elbows — pitted on pressure and was painful to touch.

Jan. 25. The animal was down and showed great pain when the swelling in the fore legs was palpated. Appetite good and she showed no distress except when compelled to move. The nose was moist, rumination active. Temperature 101.4° F. Cow slaughtered.

There was no clinical evidence of impending abortion or genital infection. On post-mortem there was evidence of a pre-existing thrombus in the left iliac artery. The genital tract was removed immediately and taken to the laboratory. On external examination it was apparently normal. The os was tightly closed by a normal cervical seal. On cutting through the uterine wall there was observed in the utero-chorionic space a moderate quantity of reddish fluid containing a few whitish flakes. The chorion was for the most part normal. The cotyledons near the cervix in the non-gravid horn separated easily. The larger and more highly functioning cotyledons on the ventral aspect of the uterus were firmly attached.

Three cotyledons showed pronounced pathological changes. One of these, a large one about ten inches from the internal os, became detached at the stem even on careful handling, leaving the firmly incarcerated cotyledon adherent to the chorion. The detached

surface of the cotyledon corresponding to the stem appeared as a blanched, sunken area, surrounded by a narrow, raised, reddened zone, irregular in outline, and about two centimeters wide. Surrounding this zone was a brownish, raised inflammatory area about four centimeters wide, thickening the chorion, and giving it a leathery consistency. There was some dry, brownish debris which could be scraped away, leaving a yellowish-brown, roughened surface beneath. The cotyledon on section seemed devoid of blood and had a whitish cooked appearance. It was softened and degenerated or necrotic.

A second cotyledon, also a large one, situated about midway between this one and the apex of the gravid horn, presented changes similar to those just described, differing but slightly in that the invasion of the chorion did not extend so far from the cotyledon.

A third cotyledon, a small one near the middle of the non-gravid horn, had evidently been detached at the stem for some time. The cotyledon itself was disorganized, and was represented by a rather dry, whitish debris, but the changes had not involved the adjacent chorionic surface. There was an irregularly round, raised, whitish surface in the uterine mucosa representing the point of attachment of the stem, but the surrounding uterine mucosa itself appeared normal.

Surrounding two other cotyledons, located together on the ventral aspect of the uterus near its center, was a gelatinous infiltration which thickened the chorion. This was most pronounced near the borders of the cotyledons and extended peripherally on an average three centimeters. The inter-cotyledonary uterine mucosa showed no evidence of inflammation, all the changes being confined to the chorion, and as far as the uterus was concerned, to the immediate point of detachment of the cotyledon.

The fetus was in good condition. The abomasum contained a straw-colored thin seromucoid material with many yellowish brown flocculi.

Inoculations from the utero-chorionic fluid, affected cotyledons, and from the lungs and digestive tract of the fetus gave pure cultures of a mould apparently identical with that injected. The cow's blood remained negative to *Bact. abortum,* and the guinea-pigs injected with extracts from the fetus, cotyledons, and utero-chorionic fluid, showed no evidence of infection with *Bact. abortum.* While abortion did not occur, uterine infection was unquestionable.

On May 23, 1924, animals No. 228 and 193 were injected with 10 cc. of a similar extract, but somewhat more diluted inasmuch as No. 88 showed the slight general reaction after the inoculation. These animals carried their calves to full term, and showed no effects of the injection.

On October 7, 1924, animals No. 247 and 250, primipara, 4 and 6 months pregnant respectively, were each injected with 10 cc. of an extract of about the same density as that given to No. 88, the first animal used. On November 1, three weeks later, No. 250

aborted. Symptoms of impending abortion had been observed for several days previous to that time. The animal was immediately slaughtered, and both uterus and fetus brought to the laboratory. The utero-chorionic fluid was increased in quantity, of a dark red color, and contained many whitish flocculi. The chorion in the gravid horn was, for the most part, firmly incarcerated, edematous, and light red in color. A few small cotyledons in the cervical end of the uterus were detached. Some of these were in the pregnant, and some in the non-pregnant horn. Others near them were easily detached. The surface of the chorion adjacent to the uterus was inflamed, roughened, and studded with numerous nodules from 1 to 4 centimeters in diameter. They were gray in color for the most part, though a few were red. These areas were devoid of epithelium, and contained dense masses of leucocytes and debris. The areas were located in patches over the entire chorion. Near the cervix they were scattered, while near the middle of the uterus and in the ovarian end of the non-pregnant horn they were more pronounced. Opposite them on the uterine mucosa were like structures. The uterine mucosa was otherwise normal except for very slight congestion in the ovarian end of the non-pregnant horn. The chorion was very extensively affected, showing cotyledons in all stages of hemorrhage and necrosis, involving even the larger and more highly functioning cotyledons. The infection appeared to start at the margins of the cotyledons, many showing a peripheral zone of hemorrhage. The chorion separated with difficulty from the more recently infected cotyledons, and in those more severely affected it was firmly incarcerated. The early lesions appeared to be hemorrhage which usually originated around the periphery of the cotyledons, gradually extending centrally to involve the entire structure. As the hemorrhage extended centrally, the chorion became firmly attached, necrosis progressed rapidly, involving the stem or point of attachment to the uterine wall, so that when the chorion was lifted, these severely affected cotyledons came away with it. The cotyledons were in all stages of hemorrhage and necrosis, but the origin and direction of the changes were quite similar in most of the cotyledons involved. Many of the cotyledons in the distal end of the non-pregnant horn were entirely detached, due to the severe necrosis involving the stems. A few in the pregnant horn were completely detached and many others came away completely when the chorion was lifted from the uterus. The severely necrosed cotyledons were much enlarged, and viewed from the uterine side of the chorion they showed a dull gray center, surrounded by areas of hemorrhage. The margins were thickened, and there was a crater-like appearance where the stem had been. We have not before observed uterine infection in which necrosis of the cotyledons was so complete immediately following parturition and in which the stems were so regularly disintegrated, leaving the cotyledons incarcerated in the chorion. For that reason we are inclined to believe that the lesions are quite characteristic for this type of infection. The

fetus was in good condition. The abomasum contained a clear mucus in which were suspended dirty straw-colored flocculi, and many dark particles resembling fecal material. The mould was recovered in pure culture from the lungs and digestive tract of this fetus. Guinea-pig inoculation failed to reveal evidence of infection with *Bacterium abortum*. The dam's blood was negative. The other animal, No. 247, is still pregnant, and due to calve in a short time.*

In all, five animals were inoculated, and of these, one aborted, and one was killed before gestation terminated. Both showed unmistakable evidence of the infection. The other three showed no ill effects whatever. While the results are not very conclusive as they relate to frequency of infection, they indicate clearly that infection of chorion and fetus by intravenous inoculation with a mould can be produced experimentally. Sections of the necrotic cotyledons, and of the uterine wall where the stems had been attached, showed lesions identical with those described by Smith. The large filaments of the organism could be seen both in the sections and scrapings from the cotyledons and the surrounding edematous areas. A culture of the mould was forwarded to Smith who kindly compared it with the one isolated by him. He found that the two strains were identical or very closely related. Intravenous inoculation of two rabbits resulted in some loss of weight, and the production of small kidney lesions similar to those observed by Smith, but somewhat less extensive.

Summing up the work briefly, we can say:

1. During the period of three years, a mould of the genus Mucor was recovered in pure culture from three aborted fetuses from the same herd.

2. Experimental inoculation of five pregnant cows with a suspension of the mould produced placental infection without abortion in one, and placental infection with abortion in another. In each of these cases a pure culture of the mould was recovered. There were negative findings in the other three.

3. The mould and associated pathological lesions were practically identical with those described by Smith.

4. As a result of the findings, we believe that a mould of the genus Mucor can be definitely added to the group of organisms which produce abortion in cattle.

* This cow calved Mar. 14th. Dystocia. Calf dead. Mould not recovered from membranes and fetus.

DESCRIPTION OF PLATES

Fig. 1. Cotyledons from Cow No. 88 showing the early stages of hemorrhage and necrosis. The chorion separated from these with difficulty. Two of the cotyledons show rounded areas of hemorrhage (A), while in the other three the hemorrhagic zone is peripheral (B). The latter types are more typical.

Fig. 2. Two of the cotyledons (from Cow No. 250) show advanced stages of hemorrhage and necrosis (A. & B). They are firmly adherent to the chorion, and easily lifted away from the uterine wall. The stems are entirely necrosed, giving a thickened crater-like appearance to the cotyledons. The third cotyledon (C) is cut in sagittal section to show the areas of hemorrhage and necrosis.

Fig. 3. End of the chorion in the non-gravid horn of Cow No. 250. The necrosis is quite well advanced and involves the inter-cotyledonous chorion. Note that the stems of all the cotyledons are necrotic, leaving the typical crater-like depressions.

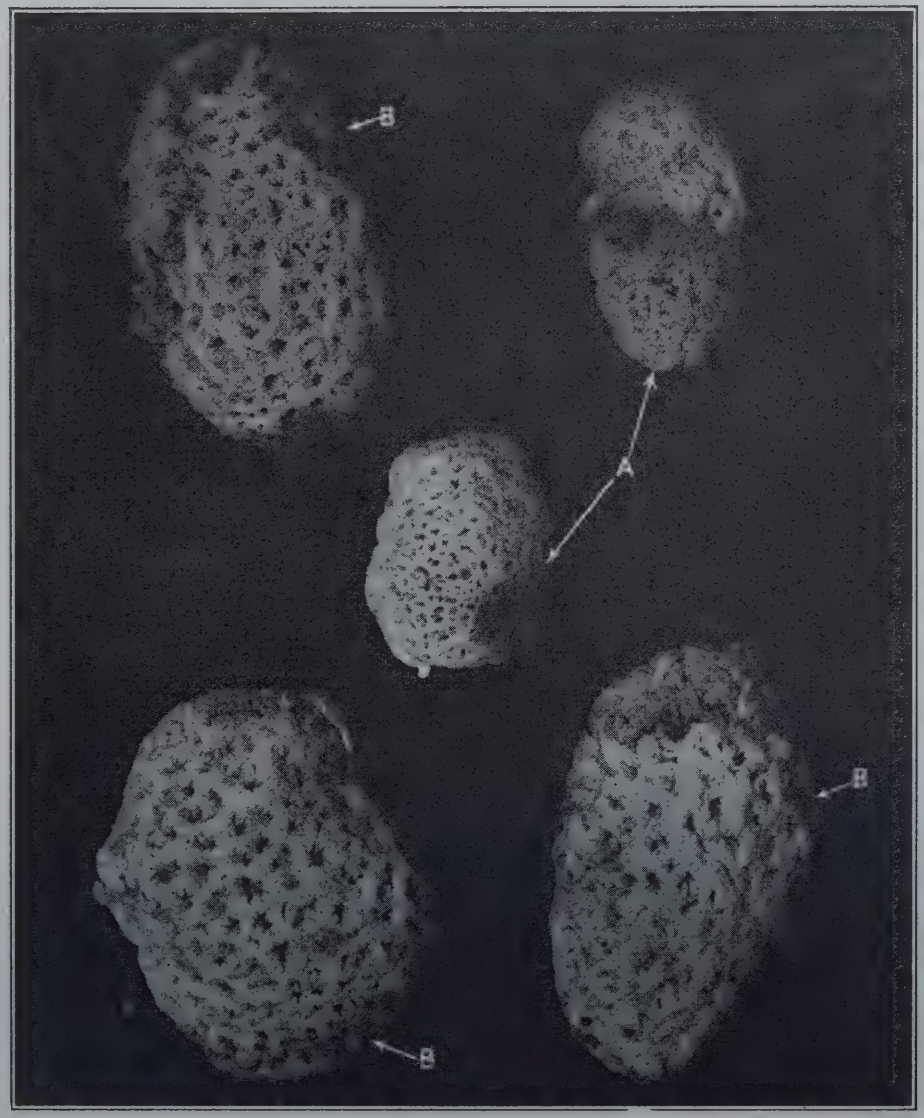


Fig. 1

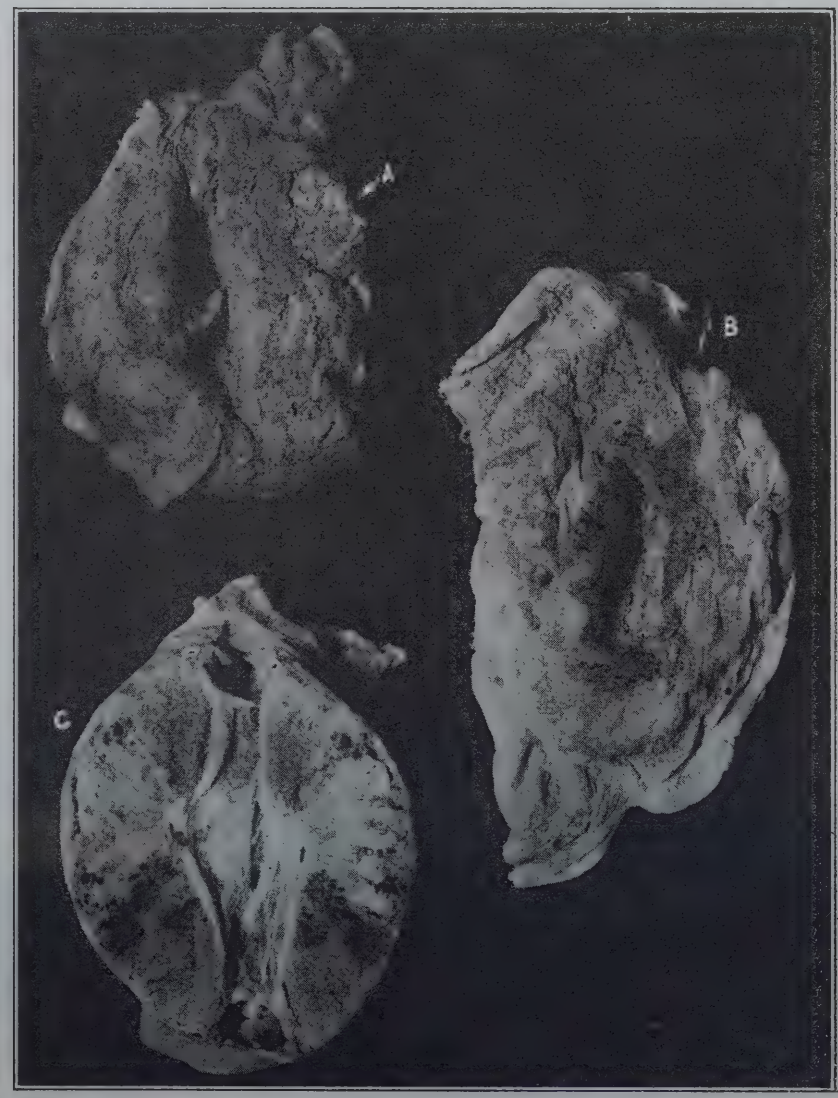


Fig. 2

Fig. 3

SOME COMMON DISEASES OF DOGS

H. J. MILKS

Department of Pharmacology and Small Animal Clinic

During the past several years we have gradually accumulated several series of interesting pictures of clinical material believing that pictures of certain subjects show more than can be brought out by description. Some of the pictures are of cases frequently seen and others of quite rare diseases.

MANGE

The first series deal with diseases of the skin and since follicular mange occurs very frequently in this vicinity, more so than sarcoptic in the dog, it will be given first place.

During the past seven years we have diagnosed 113 cases of follicular mange and our recoveries run 52 per cent. This, of course, does not mean 52 per cent of generalized cases but it does include all those upon which we were able to find the parasite; that is, every case that has been positively diagnosed as follicular mange. The parasite was found upon all cases that will be shown here.

The real purpose of showing any pictures of follicular mange or to say anything about it at this time is to try to drive home the necessity of an early diagnosis if a good proportion of recoveries is to be expected.

A dog need not have a generalized case in order to have this type of mange. A small lesion around the eyes, upon the face, around the lips or upon the paws is just as much follicular mange as the generalized or rotten cases and will become generalized soon enough if it is not handled promptly and properly. It is the early cases with which we are most often successful and but rarely in the generalized ones, although some of these surprise us and recover. This disease is no exception to the rule that we can do more for our good clients, those who consult us early, than for those who prefer to dabble in medicine and come to us as a last resort.

As you all know, there are two forms of follicular mange: the pustular which is easily recognized and the squamous which often starts with such insignificant lesions as to cause one to wonder if the trouble is really mange. In such cases the hair becomes thin or falls out leaving the parts but thinly covered or bald, smooth and with very little desquamation. Sometimes, we see the copper red color but not necessarily or usually so. In the more advanced cases the disease extends down the legs to the feet, involves the under surface of the neck or belly and may but does not always go into the pustular type.

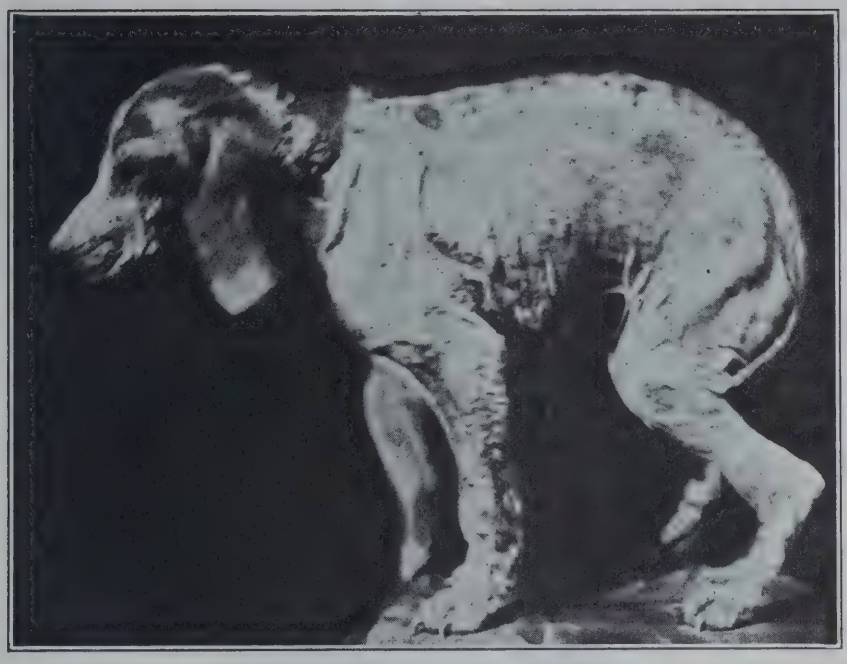


Fig. 1. A Beagle Hound with generalized case of follicular mange

Since there is little difficulty in diagnosing the pustular variety I will just touch upon the symptoms of this form. The lesions consist of dark red to bluish pustules upon the tender parts of the body; the skin becomes copper red on a dark drab scaly background. The pustules contain a sanguineous material and either rupture or are opened by the animal scratching so that in time the skin is covered with a bloody exudate. When the disease is widespread the animal smells bad and is indeed in a pitiful condition.

Fig. 2. Pustular form of follicular mange

Fig. 1 is a picture of a severe case of the squamous form that did not at any time show any pustules. The entire skin of this animal was diseased and hairless. Incidentally, it is a case that we did not cure.

Fig. 2 shows a bad case of the pustular variety of follicular mange. Note especially the thickening and folding of this skin on the face and swelling of the lower legs and feet.

Fig. 3. Boston Terrier with pustular form of follicular mange

Fig. 4. Abdomen of Boston Terrier with the pustular type of the disease

Fig. 5. Fox Hound with scattered sharply defined areas of the squamous form

Fig. 6. Boston Terrier with the squamous form around the eyes. This is a common lesion and one that responds to early treatment

Fig. 7 is sarcoptic mange in the cat and Fig. 8 the same variety in the ferret. In the latter animal the disease is often called "foot rot" although it is not confined to the feet but may involve the entire body. Note the proliferation of epidermis around the feet and the hypertrophy of the nails.

The next picture, Fig. 9, is of a rabbit with ear mange. The parasite (*Dermatocoptes cuniculi*) causing this disease is the largest of the mange mites being easily seen with the unaided eye.

Figure 10, shows an advanced case of alopecia areata. As will be seen from the picture, the dog was in good physical con-

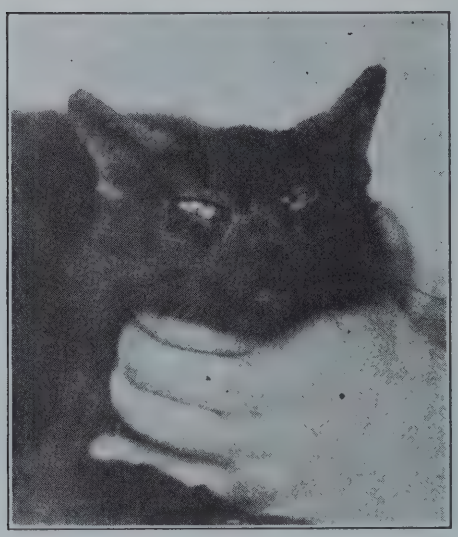


Fig. 7. Sarcoptic mange in a cat

dition. A careful physical examination was negative but the urine examination indicated that he had a mild case of diabetes. Whether this condition had anything to do with the alopecia or not could not be determined.

Fig. 8. Sarcoptic mange in a ferret

RINGWORM; CIRCINATE RINGWORM; TINEA TONSURANS

In this disease the head and extremities are most often affected although the disease may spread over the entire surface of the body. It begins with round sharply defined and single or solitary bald spots. Later, isolated clearly defined, hairless patches, round or oval in shape from ½ to 1 inch in diameter are seen upon the head, around the lips and eyes or upon the extremities or body itself. When several of these spots combine they form a larger lobulated area. The appearance varies with the stage and location

of the eruption. Frequently there are no inflammatory symptoms and there is only the baldness or the skin is covered with hair-stubs. Usually, however, the spots are covered with dirty gray or asbestos-like scales or with a thick scab which glues the lower part of the hairs together. The skin is copper red in color beneath the scabs and may be covered with numerous granules about the size of a millet seed. There are inflamed and swollen hair follicles. Occasionally the affected area projects above the surrounding

Fig. 9. Ear mange in a rabbit

Fig. 10. Alopecia areata in an Airedale.

sound skin and shows a real papular surface. The adjoining skin is swollen. The crusts may form again in some of the bald spots but in others the patches turn lighter and there is only slight desquamation. Ringworm is diagnosed by the circular shaped loss of hair extending outward, widely dispersed eruption, scurfy deposit, sharply defined spots with but slight itching, by contagiousness, and with the microscope, although it is not always easy to check a diagnosis of ringworm with the microscope.

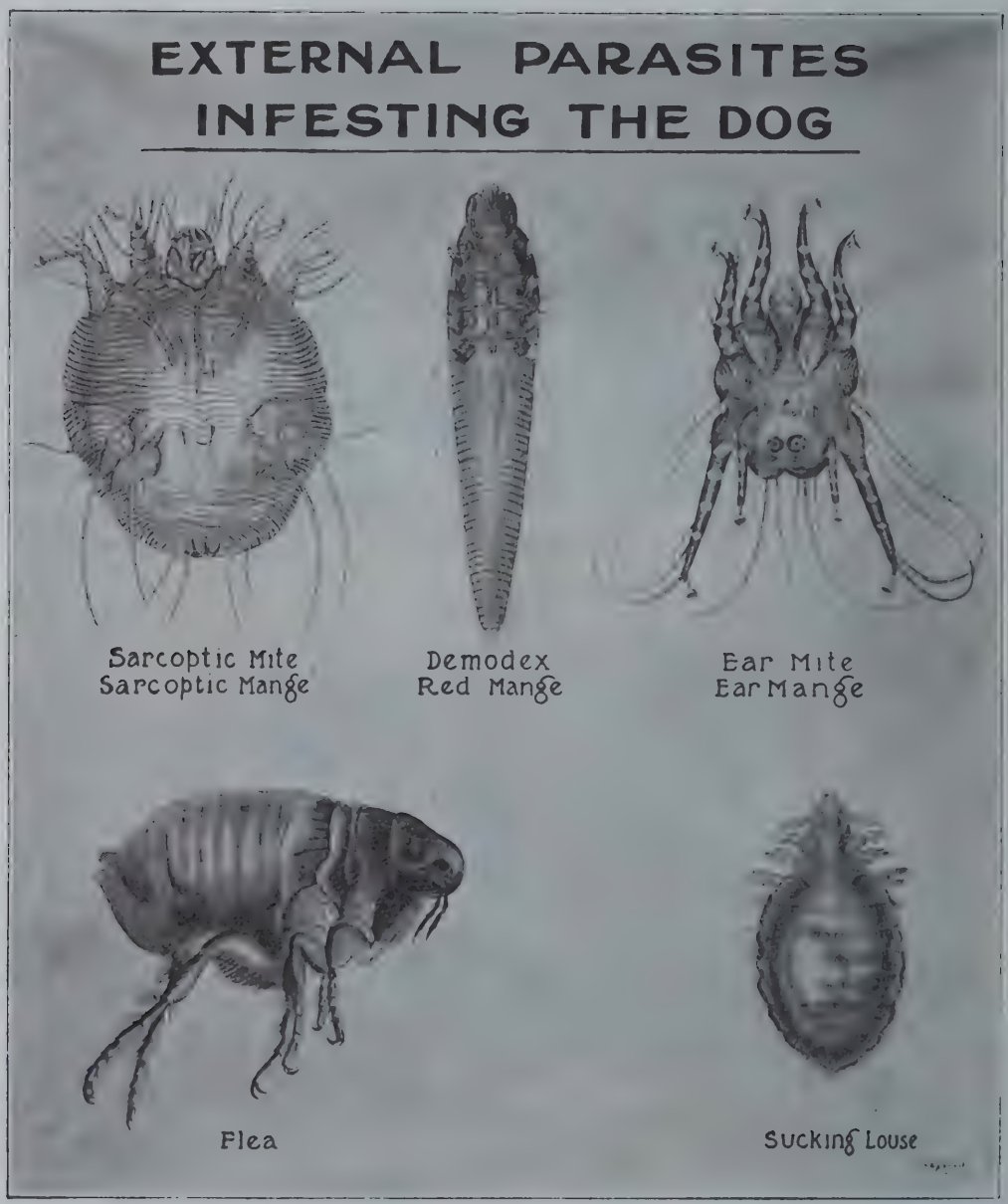

EXTERNAL PARASITES
INFESTING THE DOG
Sarcoptic Mite
Sarcoptic Mange
Demodex
Red Mange
Ear Mite
Ear Mange
Flea
Sucking Louse

Fig. 11.

Ringworm is easily transmitted to people and consequently the owner should be made acquainted with this phase of the question.

Fig. 12. Mongrel Puppy with ringworm

Fig. 13. French Bull with ringworm

ACANTHOSIS NIGRIGANS; KERATOSIS NIGRIGANS; PAPILLARY AND PIGMENTARY DYSTROPHIA

This disease is characterized by a symmetrical bilateral hypertrophy of the papillary bodies and moderate thickening of the epidermis especially in the furrows of the skin, together with an

increase of pigment in both layers of the epidermis and, unlike a similar disease in man, in the papillary bodies around the blood vessels and glands.

Acanthosis occurs in young dogs. The cause is not known. The parts most often affected are the axilla, folds of the stifle, extensor side of the toes, skin of the scrotum around the anus, under surface of the tail, lower belly, under side of the neck, corners of the mouth, eyelids and lips. The hair falls from the affected parts and the skin thickens. In the early stages it is softer than normal

Fig. 14. Acanthosis Nigrigans

and the grooves are somewhat more pronounced so that it somewhat resembles seal skin leather in appearance. (Fig. 14.) Later a bluish to black brown pigmentation takes place, elevations appear, the skin is criss-crossed with ridges or folds and is harder than normal and rough. The parts are usually itchy but there is little exfoliation.

The disease may involve the claws and mucous membranes and in case of the latter, soft, red but not bloody granulations are found on the mucous membranes.

This disease must be differentiated from follicular mange on account of the pigmentation in that disease. In follicular mange there is no symmetrical localization and the pigment may be removed with the epidermis. In addition the demodex may be demonstrated with the microscope.

Treatment. Our treatment was to soften the skin with a 5 per cent alcoholic solution of salicylic acid. This was applied every day for a week or so, then a greasy preparation was rubbed in

daily for a few days. It was found that the solution of salicylic acid loosened quite an amount of the epidermis and that this could be removed by application of the oil. The animal made good improvement and was sent home in good condition. However, the disease is prone to recur and an absolute cure rather unexpected.

Fig. 15. Genital tumors in a dog

INFECTIOUS GRANULOMA

The next series illustrates the lesions seen in the infectious venereal disease, or infectious venereal granuloma (sarcoma) of dogs. This disease affects both the dog and bitch and is characterized by soft tumors upon the genital mucous membrane. In

Fig. 16. Genital tumors in a dog.

the dog the growths appear as yellowish white raised patches varying from the size of a pin head to well defined growths on the sides of the penis (Figs. 15 and 16), or as clumps of granulating tissues at the base (Fig. 17). They bleed readily when manipulated. In the dog, the first symptom usually noticed is the bloody exudate that exudes from the prepuce, or attention is called to it by the animal's continuous licking of the part. In other cases difficulty in serving or the refusal of the dog to serve the bitch is the first symptom. In any case a protrusion and examination of the penis will clear up the diagnosis.

Fig. 17. Genital tumors in a dog

In the bitch also the first symptom noticed is a discharge of bloody exudate from the vagina. Some owners think this is a sign that she is in season and wonder why she will not breed or why she is continually in season. A digital examination of such cases will show the real state of affairs. These tumors may disappear spontaneously and when they do so may leave enough cicatricial tissues to interfere with parturition. At other times it is claimed that they may grow to sufficient size during gestation to interfere with parturition. (Fig. 18) shows an advanced case in the bitch.

These tumors are very contagious and are spread by coition from dog to bitch and vice versa. They have been responsible for serious losses in certain breeds, particularly in bull dogs. We have seen an outbreak among Beagles and individual cases in Airedales, Irish and Boston Terriers.

The treatment is removal. This may be done under either local or general anesthesia. The masses are grasped with forceps and snipped off with scissors. One must be careful to include enough of the sound mucous membrane to remove all of the tumor. The larger masses may be carefully dissected away and the sound mucous membrane brought together with a few cat gut sutures, or they may be removed by applying a litgature around their base and cutting just outside the ligature. When numerous a few may be removed at a time, allowing a sufficient interval between

operations for the mucous membrane to heal. For the very small foci the actual cautery is easy to use and very satisfactory. We have had no suitable cases for operating in the bitch.

Prevention. Since this disease is highly contagious no stud dog or brood bitch should be passed without including an examination of their genitals. We do not believe it good policy to breed a dog that has been operated upon for at least several months after operation and then only after a careful examination. Since these tumors tend to recur a better plan would be to permanently exclude all such dogs from breeding purposes.

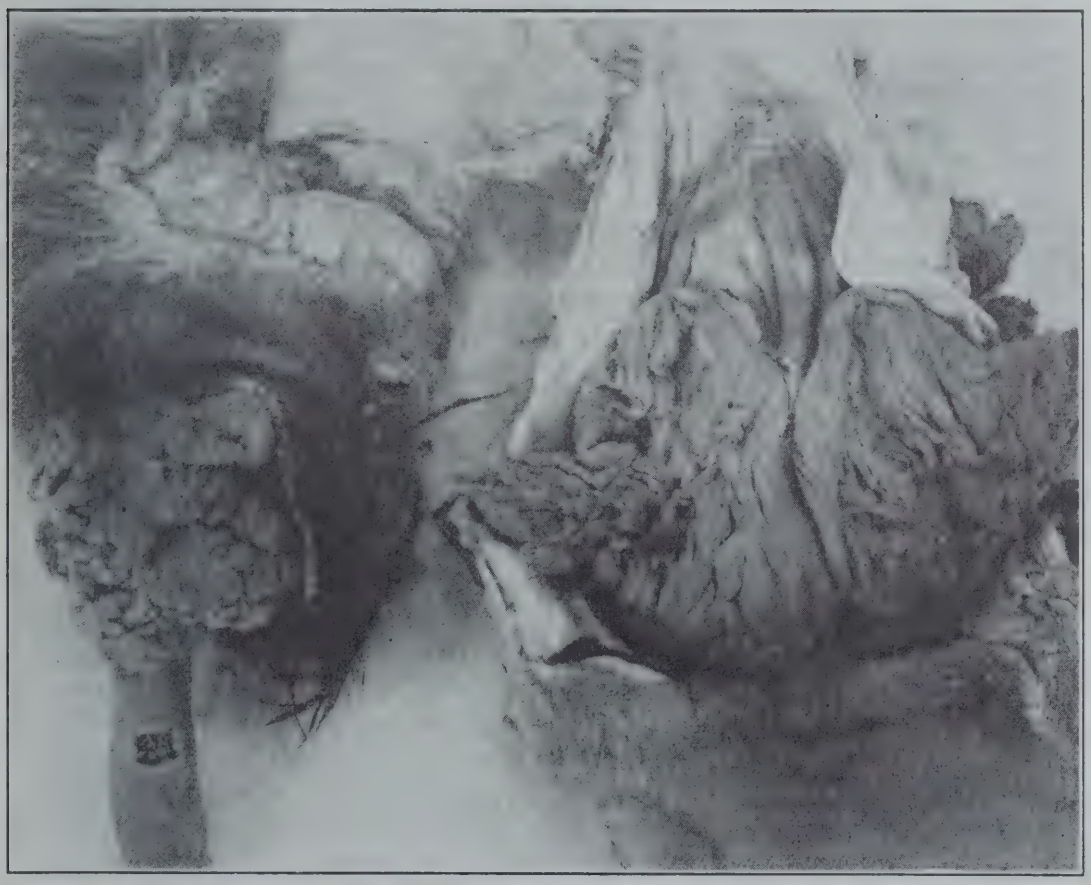


Fig. 18. Advanced cases of infectious genital tumors in the dog. Left in the dog; right in the bitch

BRACKEN POISONING OF CATTLE

W. A. HAGAN
Department of Pathology and Bacteriology

In 1920 Dr. J. K. Bosshart and I (1) described a disease of cattle which we had investigated the previous fall but were unable to diagnose. We called it a "fatal undiagnosed disease of cattle." The disease became quite prevalent in certain parts of the State during August and September and in a period of about three months resulted in the deaths of some hundreds of head of cattle. It disappeared as suddenly as it came, about the middle of November. Dr. Bosshart was sure that he had observed the disease in the autumns of 1917 and 1918, and others told me that they had known of the disease for a number of years before we began our investigation. The disease never occurred at any time other than late summer and early fall and always ceased abruptly soon after the first killing frosts.

The disease was again quite prevalent in the fall of 1920 (2). In 1921 and 1922 only a few cases are known to have occurred, but in 1923 the disease again assumed important proportions in several parts of the State. In 1924 no cases were called to our attention, but I do not doubt that it occurred as usual though probably not in serious proportions.

DESCRIPTION OF THE DISEASE

Detailed descriptions of the symptoms and pathological lesions were given in the earlier papers (1, 2) but as this was several years ago I will review some of the principal points of the disease. Only cattle are affected, particularly yearling and two-year old stock, but many cases occur in older cattle. As has been stated, the disease occurs only in the late summer and early fall. Some cases have been seen during July but the majority are seen later — in September and October. The disease appears sporadically and especially in dry years. Usually but one or two animals become affected in any one herd, either simultaneously or a few days apart, but when the pastures are exceedingly dry many cases may occur in a single herd. Many outbreaks occur in very isolated farms on which there is no history of previous cases and to which, because of their isolated position, it is difficult to see how infection could have been brought. I have known of the disease occurring in single animals in herds which were removed many miles from any other known cases.

Most of the cattle show an acute disease. An extremely high temperature is an early and significant symptom. This usually exceeds 106° F. and not infrequently is above 108° and even 109°. The affected animals stand with their heads down and drool from the mouth. A thin bloody fluid sometimes trickles from the nostrils. Nearly always careful search will reveal petechiation of the visible mucosae. If the animal lives longer than twenty-four

hours after the onset of the symptoms, blood usually will be passed with the feces either as black masses or as bright red clots. The animals usually die within one or two days.

The principal lesions are of a hemorrhagic nature. Petechiae and larger hemorrhages are found in nearly all parts of the animal, being especially noticeable in the subcutaneous tissue and on the serous membrane. Striking lesions are also found in the digestive canal, and in the liver. The abomasal wall shows edema of the mucosa and numerous hemorrhages, particularly about the duodenal opening. Some of the hemorrhages appear to be several days old; the membrane over the center of the hemorrhagic areas has ulcerated. Similar hemorrhages and sometimes ulcers are found commonly throughout the small intestine. The content of the small intestine usually is mixed with more or less blood which apparently has come from the shallow ulcers. In many cases the cecum and large intestine are filled with a single large mass of clotted blood of a bright red color, in other cases there are many smaller clots mixed with the ingesta. Characteristic necrotic areas are found nearly always in the liver. These may easily be mistaken for those of the "necrosis bacillus" but careful examinations have shown that they are not due to this organism. Inside the necrotic areas numerous bacteria are found in clumps and huge masses. The size and position of these masses suggest that the bacteria have multiplied in the lesion after it has been formed.

Bacteriological work on the disease — that already reported upon and other since carried out — has proven fruitless. The organs, with the exception of the necrotic areas of the liver, are usually sterile and the liver lesions have not yielded any organisms of significance. All inoculation experiments in small animals and cattle have resulted in failure to infect. The principal point established by the bacteriological work is that the disease is neither anthrax nor hemorrhagic septicemia, the two guises under which the disease has generally been parading, indeed the work has even failed to show that the trouble is infectious. We stated in the paper in 1920: "All efforts that have been made toward a diagnosis of this disease have failed. We have regarded it as infectious, but the possibility of some kind of plant or fungus poisoning has not been lost sight of. There are no lesions but what might conceivably be produced by a toxic substance derived from plants." The assistance of a botanist was sought to inspect the vegetation of pastures in which the disease had occurred that known poisonous plants might be detected. His report was negative; he found nothing which in his opinion might be incriminated.

In the late summer of 1923, I was called to Chautauqua county to investigate a disease of cattle which had alarmed the farmers in the vicinity of Clymer. A number of cattle had died. The disease was diagnosed as anthrax by one veterinarian and the cattle had been vaccinated with the spore vaccine. When they continued to die another veterinarian was called in, gave a diagnosis of hemorrhagic septicemia and administered hemorrhagic septicemia

bacterin. When the animals continued to die an investigation was solicited. The disease proved to be the one under discussion. Typical clinical symptoms and autopsy findings were obtained on two farms a number of miles apart.

While looking over the pastures where the affected animals had been grazing it was noticed that the many ferns (ordinary bracken, brake-ferns, *Pteris aquilina*) had been pretty well grazed down by the cattle in both places. These plants were practically the only green vegetation left in the pastures since the season was exceedingly dry and everything else was badly burned. Since cattle do not usually eat this plant, at least to any considerable extent, the idea suggested itself that there, perhaps, was the cause of the trouble, that some of the cattle had been driven by pasture shortage to eat this plant which usually they ignored and that poisoning had resulted from it. Arrangements were made, therefore, to have a quantity of the brakes cut in the pastures where the disease had occurred and shipped to the laboratory in Ithaca where a feeding experiment was started.

Two cows were secured for the experiment. They at first refused the brakes, but when other food was withheld they each finally consumed about twenty pounds of the partially dried plants. When a number of days had elapsed with no sign of illness occurring, a second shipment of brakes was fed as before. One animal developed mastitis and showed fever and other symptoms referable to this infection; the other remained well thruout the period of observation. At the end of five weeks from the time of the first feeding both animals were slaughtered. They showed no lesions which could be associated with the feeding of the brakes.

Altho my findings, as a result of the single feeding experiment just described, failed to indicate any poisonous property for cattle in the common brake, a subsequent investigation of some of the literature has brought to light some facts which are pertinent to this question. Pammel (3) merely states that the plant "is suspected of being poisonous." Hadwen (4) showed clearly that dried brakes are poisonous for horses; that continued feeding of hay containing a considerable proportion of brakes results after about one month in the development of "staggers."

Poisoning of cattle thru the eating of bracken appears to have been described first by Storrar (5). The outbreak occurred in Great Britain in 1893. It was in midsummer of an exceedingly dry season. The pasture on which the herd was grazing was "burnt up" with the exception of ferns which were abundant. The young fronds of these ferns had been eaten off closely. There is hardly any need to describe the symptoms found in the cattle which became affected with the disease and died, for they were identical with those of the disease which Bosshart and I described in 1920. There was an extremely high temperature, cessation of milk supply, frequently a thin bloody nasal discharge, drooling from the mouth, blood in the feces and death within a day or two. Storrar remarks particularly about the extremely high temperature, saying,

"The temperature is characteristic and in my opinion quite enough to indicate the disease, as I do not know any other where the temperature runs up so high in the first stages, and while many of the symptoms which I have described, are not yet apparent." The autopsy findings reported by Storrar are identical with those found by us except that he does not mention the finding of the liver lesions. It seems rather improbable that a close observer as Storrar appears to be could have overlooked such evident lesions, and as later writers make no mention of the lesions in their descriptions, I assume they are not present in the disease which occurs in Great Britain.

Stockman (6) described a disease of cattle, which apparently is quite well known in England, under the name of bracken poisoning. The serious outbreaks of this disease occur there exclusively in the autumn months—from August to the beginning of November. The symptoms and lesions are the same as those described by Storrar. The disease is one of cattle at pasture and always associated with the eating of brakes. To prove that the brakes were responsible for the disease Stockman undertook feeding experiments with two head of cattle. Thinking that if the brakes were poisonous one or at least a few large feedings ought to give results, he administered a total of about 60 lbs. of the freshly cut plants to each of two animals over a period of about one week. Except for slight symptoms of indigestion the results of the experiment were negative.

In the meantime Stockman was called in to investigate a fatal disease in cattle which were being fed upon a soy bean by-product, a meal which had been defatted with trichlorethylene. Upon this material the cattle appeared to thrive for a considerable period of time but eventually many of them developed symptoms of acute poisoning and died. The symptoms and lesions in these animals were identical with those which were commonly found in the disease known, in Great Britain, as bracken poisoning. From this experience Stockman concluded that there was some sort of plant poison, probably the same in the soy bean as in bracken, which "acted slowly, either by accumulation, protracted action or both, finally and suddenly producing in an explosive manner a severe and often fatal illness, characterized by blood destruction, general hemorrhages and nervous symptoms, and that poisoning was invariably ushered in in its acute stage by a high temperature, as in the case of bacterial diseases."

In the light of the new knowledge a second feeding with bracken was now undertaken by Stockman. An eight-months old bull calf which had never had any bracken, was fed 10 pounds of cut brake shoots every day, together with other food. The animal thrived until the 26th day of the feeding when he was found to be ill, with a temperature of 106°F. He was dead on the fourth morning of the illness. The clinical symptoms and lesions were characteristic in every way of the naturally occurring disease known as bracken poisoning.

DISCUSSION

I do not have the data as yet to make a definite diagnosis of bracken poisoning upon the New York cattle disease. Such a diagnosis can be made only after conclusive feeding experiments upon normal cattle. We have plans laid and hope to make such an experiment this summer, but the results will probably be too late for practical application to the field cases which will surely develop this summer and fall. For this reason, I have laid the circumstantial evidence before you in this preliminary report that you may be on your guard when the disease recurs and may possibly be able to save some stock which otherwise might perish.

The circumstances of its development, its symptoms and lesions are so nearly like those of the English disease that there can hardly be any doubt about the identity of the causative agents, whatever they may be. In neither the work abroad or that done here was any evidence of a microörganismal cause for the disease discovered, and although this should not be taken as definite evidence of the absence of such a cause, there are other evidences which point away from infection and toward plant poisoning, as the cause of the disease. I would sum these up as follows:

1. The disease never occurs except in the latter part of the growing season, and when the animals are in pasture. Cases may develop in animals which have been removed from pastures for several weeks, but these may be explained by the cumulative action which was suggested by Stockman.

2. I have noted several instances in this country and several have been noted in England of outbreaks among cattle which have fed on brakes growing in pastures in which nearly all other vegetation has disappeared because of dry, hot weather.

3. The cases cease rather abruptly in the fall after the vegetation is frosted.

4. Stockman has produced a typical case of the disease in a normal animal by feeding a moderate amount of brakes daily over a period of 26 days. Hadwen has produced disease in horses tho of entirely different type by continuous feeding of dried brakes in hay.

Many will probably be skeptical of the fact that the ordinary brake can be the cause of poisoning in view of the almost universal presence of the plant in pastures in this state and from the fact that cattle are seen not infrequently to nibble at the young fronds, but we have already explained that to produce poisoning it is necessary that a considerable amount of the fresh plant be consumed regularly over a considerable period of time, and cattle are not sufficiently fond of the plant to do this under ordinary circumstances. It can easily be understood how poisoning may occur when the pastures are poor, but the disease also frequently occurs when the pastures are relatively good, though I have not known of any instances when many cattle have been lost in any one herd under these conditions. It is assumed in this case that a few animals develop a taste for the bracken and as the pastures become

shorter as midsummer approaches they consume more until poisoning becomes manifest. It is possible also that the poisonous principle does not develop in the plant until it is fairly mature, as is the case with certain alkaloids.

In view of the possibility or even probability that poisoning by brakes explains the annual losses which have been occurring regularly each fall in many parts of this state, I would suggest that when the disease is encountered the cattle be immediately ordered out of the brakey pasture, and all animals be given a saline cathartic. Some deaths within a week or two after removal from the pasture should not be unexpected. Cutting or destroying the brakes should be done if it is feasible.

BIBLIOGRAPHY.

1. BOSSHART, J. K. and HAGAN, W. A., *Cornell Vet.*, 1920, x, 102.
2. HAGAN, W. A., *Cornell Vet.*, 1922, xii, 43.
3. PAMMEL, L. H., *A Manual of Poisonous Plants*, 1911, The Torch Press, Cedar Rapids, Iowa.
4. HADWEN, S., *J. A. V. M. A.*, 1917, x, 702.
5. STORRAR, D. M., *J. Comp. Path. and Therap.*, 1893, vi, 276.
6. STOCKMAN, *J. Comp. Path. and Therap.*, 1917, xxx, 311.

THE GREEN COLORATION PRODUCED BY CERTAIN STREPTOCOCCI ON BLOOD AGAR PLATES*

WILLIAM A. HAGAN

Department of Pathology and Bacteriology

McLeod, Gordon and Pyrah (7) showed that certain peroxide forming bacteria produced a greenish discoloration when they were grown in a cooked blood (chocolate) medium. This suggested the idea that in peroxide formation probably lay the explanation for the formation of the green coloration which appears about colonies of pneumococci and certain streptococci when they are growing on blood agar plates. Since no satisfactory explantion of the mechanism of this phenomenon, which has long been used as an important criterion in the identification of organisms of these groups, had ever been offered, some experiments toward this end were undertaken.

Ruediger (10) was led to believe that the formation of a green discoloration around colonies of certain cocci growing on blood agar was due to acid formation. Certain streptococci which hemolyzed blood cells suspended in plain agar produced green when they were grown in the presence of blood on dextrose agar. On the other hand, pneumococci which produce a deep green color when grown in plain infusion or dextrose agar, produced only a faint trace of green when they were grown in a blood medium with a peptone water base. Ruediger's explanation does not take into account the zone of hemolysis which regularly occurs at the periphery of the zone of discolored cells.

Mandelbaum (8) assumed that *Streptococcus mitior* produced two substances which acted upon the blood cells. One, his "hemaglobin-toxin," acted upon the blood cells changing the color of the hemoglobin and rendering the cells insusceptible to the second substance, his "hematoxin," which hemolyzed normal cells. The first substance was supposed to be closely bound up with the living bacterial cells hence it affected only those blood cells which lay in immediate contact with the colony. This explanation accounted very well for the presence of a group of discolored cells lying immediately around the colony when cells farther away were hemolyzed, but it cannot explain the phenomenon described by Brown (4) who found that by changing the temperature while the colony was growing multiple zones of discolored cells separated by zones of hemolysis could be produced.

Brown suggested an explanation which fitted in well with all observed facts, and with the new facts given in this paper. His explanation is given in his own words:

"It may be assumed that there are elaborated by the *Streptococcus mitior* colony two substances, one of which (a) produces hemolysis and the other of which (b) produces changes in the

* Jour. Inf. Dis., 1925, xxxvii, p. 1.

corpuscles visible as a greenish or a brownish green discoloration of the intact corpuscles rendering them resistant to hemolysis by the first substance. It must be assumed that in the development of the colony the second substance (b) is elaborated first or at any rate reaches a concentration sufficient to produce fixation of the corpuscles next to the colony before the hemolytic substance (a) is present in sufficient concentration to produce hemolysis. The hemolytic substance acts well at refrigerator temperature, but at temperatures much below that of the incubator the fixing substance (b) is not produced or is inactive."

Butterfield and Peabody (5) showed that the oxygen capacity of blood cells which had been incubated with pneumococcus cultures was markedly reduced and by means of the spectroscope demonstrated that the reason for this was that much of the hemoglobin had been changed to the form of methemoglobin. Cole (6) confirmed and extended the observations of these workers. He concluded that the formation of methemoglobin by the pneumococcus was an oxidative process, or possibly a mixture of oxidizing and reducing processes. Blake (3) concluded that the green color produced by streptococci of the viridans group was due to the formation of methemoglobin, but states that "the exact chemical change is unknown."

EXPERIMENTAL

All of the cultural work reported in this paper was done with a single strain of a green producing streptococcus which had been isolated from a normal throat. It was a typical alpha type Smith and Brown (12), or viridans type Scotmüller (11). It grew rapidly in infusion broth, producing marked turbidity in twelve hours or less at 37°C. Growth occurred, but less rapidly, at room temperature.

In blood agar plates (10 to 12 cc. infusion agar, pH 7.4 plus 0.6 cc. defibrinated horse blood) strong green pigmentation of colonies was evident after 24 hours' incubation at body temperature. When plates were incubated for 48 hours or longer at this temperature an indefinite halo appeared around the colonies at the periphery of the zone of discolored cells. (Fig. 1.) This was due to hemolysis of a portion of the blood cells which lay in this region. When the suggestion of Brown (4) that the plates be placed in the refrigerator for 24 hours after being incubated for one or two days was followed, a sharply demarcated zone at the periphery of the discolored area always appeared. The cells in this zone were completely, or nearly completely, hemolyzed. (Fig. 2.) It was noted that the peripheral hemolysis always appeared earlier and was more conspicuous in incubated plates when large numbers of colonies were present on the plate (previously noted by Smith and Brown), and in parts where the agar layer was thin. Hemolysis was also noted more frequently when the proportion of blood to agar was lower (previously noted by Rieke, 9). In the plates where the colonies were numerous, the entire mass of blood medium was altered in a greater or lesser

degree, the normal blood color being changed to brownish and greenish-brown tones.

When infusion agar plates were incubated at 20-25°C. instead of at body temperature the effect upon the blood medium was quite different. The colonies, instead of being surrounded by masses of deeply green tinged cells, showed only traces of green, and hemolysis was more marked. When the colonies had reached their maximum development after four or five days' incubation, they frequently presented an appearance very much like that of hemolytic streptococci (beta type, Smith and Brown). Microscopic examination always showed, however, that there were a few pale green tinged, intact corpuscles in the immediate vicinity of the colony, and the hemolyzed zone itself usually showed a few intact cells, so that it was not so clear as that of the true hemolytic streptococcus. The appearance was very much like that described by Brown for streptococci of his alpha prime group. (Fig. 4.)

When one per cent dextrose was added to infusion agar as a basis for blood agar plates, the appearance of the colonies was changed greatly. The most heavily seeded plates became opaque grayish brown after 24 hours' incubation. In the less crowded plates the colonies developed exceedingly rapidly. At first green discoloration of the cells around the colonies could be seen but by the time the colonies were 24 hours old much of the green color had been replaced by a brownish-black. (Fig. 3.) Practically all of the corpuscles lying in the medium between the colonies were hemolyzed. The medium became perfectly clear but of a light brown color.

On an extract agar base the colonies always were small and green production was slight. Hemolysis was always a prominent feature. The appearance was much like that of colonies of the same organism growing upon infusion-blood-agar plates but at room temperature.

When infusion-blood-agar plates were incubated in a Brown anaerobic jar, or in a sealed jar with alkaline pyrogallol, growth always was tardy and the colonies small. Green production was always slight but perceptible when the jars were incubated immediately after they had been sealed. When they were held in the refrigerator for 24 hours before incubation so that growth of the organism was restrained until anaerobic conditions had been fairly established and the color of the blood had turned to the "burgundy red" of reduced hemoglobin, green formation never occurred. When the jars were opened after incubation and access of the air was allowed, green formation became evident around the colonies after a few minutes.

PRODUCTION OF PEROXIDE BY STREPTOCOCCUS VIRIDANS

The streptococcus used in this work was tested for its ability to produce peroxide by the method suggested by Avery and Morgan (1). It proved to be a vigorous producer of peroxide when

conditions were favorable for active growth in the presence of abundant free oxygen. Cultures grown in very shallow layers of infusion broth at 37°C. gave positive tests for the presence of peroxide in a few hours — generally after eight or ten hours' incubation very strongly positive tests were obtained. When cultures were grown in tall columns of fluid with comparatively little surface, or if the surface was covered with vaseline, tests for peroxide were always negative after 12 to 24 hours incubation, but when the cultures were then poured into flasks so as to make a shallow layer of fluid freely exposed to the air, positive tests for peroxide could always be obtained in one or two hours at body temperature.

Effect of Age of Culture upon Its Ability to Produce Peroxide

When cultures were grown under a vaseline seal in fluid media and subsequently exposed to the air by pouring them into large flasks, it was soon noted that the rapidity of peroxide accumulation, if, indeed, it accumulated at all, depended upon the stage of growth of the culture at the time it was exposed. Infusion broth cultures seemed to be in best condition for accumulating peroxide quickly when they were from 12 to 24 hours old. Dextrose broth cultures at 24 hours frequently failed to produce peroxide when exposed — the optimum time for this medium appeared to be less than 12 hours. Extract broth supported growth poorly and peroxide formation was always slight in this medium, and rather slow to develop. Cultures in plain infusion, dextrose infusion, and extract broths which had been incubated under vaseline seal for one week failed to accumulate sufficient peroxide when they were exposed to the air to give a positive reaction to the test reagents.

Effect of Temperature upon the Rate of Peroxide Accumulation by *Streptococcus viridans*

The rapidity of peroxide accumulation by cultures exposed to the air is markedly influenced by the temperature at which they are exposed. This is illustrated by the following experiment: The strain was grown in plain infusion broth, pH 7.4 for 24 hours at 37°C. under a vaseline seal. The culture was then divided into three equal portions, one part being placed in each of three 250 cc. flasks, the fluid forming a shallow layer in the bottoms of the flasks. One flask was held at incubator temperature, the second at room temperature, and the third was refrigerated. As a result of periodic tests, peroxide was detected in the first flask after two hours, and in the second after six hours. Repeated tests up to the 96th hour failed to demonstrate peroxide in the third flask. At this time the flask was placed in the incubator. A faintly positive test for peroxide was obtained after three hours and after six hours a strongly postive test was obtained.

EFFECT OF COMPOSITION OF THE MEDIUM UPON PEROXIDE FORMATION BY STREPTOCOCCUS VIRIDANS

A ten hour culture of the organism in dextrose infusion broth grown under a vaseline seal, was thoroughly centrifuged to throw down the bacteria. The sedimented bacteria were suspended in a small amount of sterile physiological salt solution. One cc. portions of this suspension were then placed in 10 cc. lots of culture fluids of varying composition and reaction. The amount of culture added was sufficient to make a very heavy suspension. The various media were then poured into large flasks so as to expose the fluids freely to the air. The flasks were incubated at 37°C. Tests for peroxide formation were made at intervals. The details are given in Table I.

TABLE I

Effect of Composition and Reaction of Medium upon the Rate of Peroxide Accumulation by *Streptococcus viridans.*

The symbols represent the comparative intensities of the tests obtained for the presence of peroxide.

	Immediately	1 hr.	2 hrs.	4 hrs.	9 hrs.	24 hrs.
Extract broth pH 5.0	—	—	—	—	—	—
Extract broth pH 6.0	—	—	—	—	—	2+
Extract broth pH 7.0	—	+	+	2+	4+	3+
Extract broth pH 8.0	—	—	+	2+	4+	3+
Infusion broth pH 5.0	—	+—	2+	2+	—	—
Infusion broth pH 6.0	—	+	3+	6+	10+	5+
Infusion broth pH 7.0	—	3+	5+	7+	10+	8+
Infusion broth pH 8.0	—	2+	4+	6+	8+	5+
Dextrose infusion broth pH 5.0	—	—	+	3+	4+	+
Dextrose infusion broth pH 6.0	—	+—	3+	4+	10+	4+
Dextrose infusion broth pH 7.0	—	+	3+	4+	6+	3+
Dextrose infusion broth pH 8.0	—	—	+—	2+	5+	+—
Infusion broth pH 7.0 plus 0.1% formalin	—	—	—	—	—	—
Infusion broth pH 7.0 plus 0.5% phenol	—	—	—	—	—	—
Infusion broth pH 7.0 plus 0.1% phenol	—	+—	2+	6+	8+	4+
Infusion broth pH 7.0 heated for 20 minutes at 60° C after the addition of the culture	—	—	—	—	—	—
Physiological salt solution	—	—	—	—	—	—

Inspection of the data in Table 1 brings out the fact that peroxide is produced most rapidly and in greatest abundance in those fluids in which the composition and reaction favored the growth of the organisms which were suspended in them. Although the quantity of bacterial cells added to each of the fluids was greater than would have developed in the media under optimum conditions, yet no detectable peroxide was formed in those fluids which were not favorable for the vital activities of the bacterial cells which were added.

The conclusions drawn from the experimental evidence relating to the production of peroxide by the strain of streptococcus studied are:

1. Peroxide formation occurs in cultures when they are held under suitable conditions. The necessary conditions are:

a. Free oxygen must be present in sufficient amount. This requirement may be met by cultivating or exposing the cultures in shallow layers of fluid media in free contact with air. Peroxide is not produced, at least not in sufficient quantity to be detected by the methods used, in cultures grown in the absence of air.

b. The organisms must be living and under conditions which permit of active exercise of their metabolic processes. Organisms killed with heat and chemicals do not form peroxide. Living organisms held under conditions which do not permit of active multiplication, that is, when they are suspended in physiological salt solution, when they are suspended in fluids saturated with their own products of growth (old cultures), when they are suspended in media of unsuitable composition, and when they are suspended in media of suitable composition but at a temperature too low for active multiplication, do not produce peroxide.

These conclusions are somewhat at variance with those of Avery and Neill (2) who found that whole cultures of pneumococcus held under conditions not permitting of active growth, and even sterile extracts of pneumococcus cultures, were capable of producing peroxide when they were exposed to air.

2. Peroxide is produced most rapidly and, consequently, accumulates in greatest amount in those media which allow of the most rapid growth. In extract medium, a rather unfavorable substrate for this organism, peroxide always accumulated slowly and in low concentration.

3. Peroxide is produced in greatest amount during the period of logarithmic increase. Cultures incubated anaerobically until this period has passed show little or no tendency to accumulate peroxide and in cultures which have been allowed to form peroxide during the logarithmic phase of growth, the concentration rapidly decreases after this period has passed.

Action of Peroxide and of Acids upon Blood Agar

It seemed that, if the green coloration which appeared around the colonies of certain microörganisms when they were growing in blood medium was due to the action upon the blood of peroxide produced by the organisms, it should be possible to produce this color artificially. With this idea in mind, solutions of peroxide of varying strength were dropped on the surfaces of blood plates. The stronger solutions showed strong effervescence and the agar frequently was fractured by the violence of the gas liberated but in no case was there any discoloration of the corpuscles.

Very interesting results were obtained when small drops of acid solutions were placed on the surfaces of sterile blood plates. Sulphuric, hydrochloric, acetic, butyric and lactic acids gave identical results hence it appears that the phenomena are due to the activities of the hydrogen ions alone.

When the acid was in concentrated solution the medium immediately beneath the drop promptly became grayish-brown in color and somewhat translucent. Within a few minutes the translucency gave way to opacity and the color changed to a grayish-green. Around the periphery of the opaque area a somewhat translucent zone began to become apparent after a few minutes and after some hours this region was perfectly transparent because of complete hemolysis of the corpuscles. (Figs. 5 and 7.) The width of this zone varied according to the nature of the medium in which the blood cells were suspended, being widest in extract agar (low buffer content) and narrowest in infusion agar which had been buffered highly with a phosphate mixture. When a very small amount of acid solution was introduced into the agar layer by stabbing it with a very fine pointed pipette, the small group of discolored cells surrounded by a completely hemolyzed zone which resulted, simulated so closely the appearance of a colony of a green producing streptococcus which had been refrigerated after incubation that it would easily deceive a casual observer examining the "colony" without magnification. (Fig. 6.) The green color was lacking, however, the discolored cells in this case being greenish gray instead of the characteristic bottle green of the true colony.

When more dilute acids were used, the area beneath the drop became transparent more slowly and the final opacity was less but the character of the hemolyzed zone about the central opacity remained the same. When still weaker solutions were used, a concentration could be found which gave a completely hemolyzed area beneath the drop without the development of a central opacity (2%–3% lactic acid upon infusion medium; less than 1% lactic acid upon extract medium).

In this case there was little immediate change in the medium beneath the drop but gradually transparency developed as the hemolysis progressed. Usually six to eight hours were required for hemolysis to become complete.

Acid solutions which were slightly too weak to produce hemolysis caused the red color of the blood to change to a reddish-brown color. This color was also seen frequently around the periphery of the zone of hemolyzed cells when stronger acids were used. The color is the same as that seen in plate cultures of *Streptococcus viridans* when the cultures are crowded.

It was decided to ascertain the combined effect of acid and peroxide upon blood agar. Accordingly a series of dilutions of lactic acid were made up and after a drop of each dilution had been placed upon the surface of a blood agar plate, approximately one part in five thousand of commercial hydrogen peroxide was added to each of the solutions and a second series of drops was made on the same plate. The effect produced by the acids containing a trace of peroxide was strikingly different than when acid alone was used. The differences were: (1) strong acid plus peroxide produced opaque deep green zones instead of the green-

ish-gray of acid alone and (2) the peroxide exerted a distinct protective action on the corpuscles when in hemolyzing concentrations of acid. This action was evidenced when the stronger acid solutions were used by the absence of the initial stage of translucency, but it was most strikingly seen in concentrations of acid which were just capable of completely hemolyzing the corpuscles. Hemolysis in this case was always completely inhibited and instead the blood cells were given a light green tinge. (Figs. 8 and 9.)

THE MECHANISM OF GREEN PRODUCTION BY STREPTOCOCCUS VIRIDANS WHEN GROWING IN BLOOD AGAR

The experimental evidence given in this paper leads to the conclusion that the green pigmentation produced by certain streptococci in blood agar plate cultures is due to the action upon the blood corpuscles of peroxide, probably hydrogen peroxide, in the presence of acid. Both of these substances are produced simultaneously by the organism and diffuse outward from the colonies thus bringing about changes of the blood in the immediate vicinity of the colony. The evidence for this hypothesis is as follows:

1. Acid alone does not account for the phenomenon. Strong acids alone produce a greenish-gray discoloration, but not the bottle green which is characteristic of the *Streptococcus viridans* colony.

2. Green producing organisms are peroxide producers, but peroxide, or hydrogen peroxide, at least, does not cause discoloration of blood corpuscles.

3. Hydrogen peroxide, in the presence of a suitable acid concentration, has been shown to produce the characteristic bottle green color in blood corpuscles. A low concentration of hydrogen peroxide in sterile broth of a reaction of pH 7.0 will not discolor a small amount of blood corpuscles added to it, but the same concentration in broth of a reaction of pH 4.5 will cause brownish-green discoloration.

4. When precautions are taken to prevent the formation of peroxide by organisms growing on blood agar plates by incubating them under strictly anaerobic conditions, green formation does not occur. When access to air is allowed to these plates, the green discoloration rapidly appears around the colonies.

5. Green production is slight when the organism is cultivated upon blood plates made with an extract agar base, and strong when made with infusion agar. It has been shown experimentally that little peroxide is formed when the organism is growing in the first medium while the latter is quite favorable for its production.

6. Green production is slight when the organism is cultivated upon infusion agar blood plates at room temperature. It has been shown that peroxide formation proceeds very slowly when organisms are cultivated at this temperature.

THE MECHANISM OF HEMOLYSIS BY STREPTOCOCCUS VIRIDANS

The hemolysis produced by streptococci of the green producing group after long incubation or, better, by refrigerating the plate cultures after a period of incubation, seems capable of adequate explanation upon the basis of acid production alone. This fact is suggested by the behavior of acid solutions upon blood agar, but other evidence is at hand.

1. Hemolysis is always most marked in plates made with a basic medium of low buffer content (extract agar).

2. It has been shown that acid hemolysis is restrained by the presence of peroxide. Whenever peroxide formation is inhibited by placing the organisms under conditions where growth is less rapid (lower temperature of incubation, composition of medium less suited for growth) hemolysis becomes a prominent feature of the colony characteristics.

3. Hemolysis in cultures growing at body temperature is always a belated phenomenon. It has been shown that peroxide formation which goes on rapidly during the early hours of growth soon ceases or diminishes, hence it would be expected that its restraining influence on hemolysis would be removed after the period of rapid growth had ceased. Complete suppression of peroxide production in young cultures by refrigeration thus leaving the acid free to cause hemolysis would also adequately explain the facts observed. Peroxide quickly disappears when it ceases to be produced, but acid will continue to diffuse.

4. Hemolysis is always more marked in crowded plate cultures where it is evident that the colonies exert a mutual effect upon each other. The general alteration of the color of the hemoglobin to a brownish cast is evidence of the general diffusion of acid throughout the medium. In this case conditions are favorable for the more rapid accumulation of a hemolyzing concentration of acid around the colonies and the inhibiting effect of crowding causes peroxide formation to be curtailed early. Less opportunity for the diffusion of the acids is found on plates where the agar layer is thin, and when fewer blood cells are present in the medium. the smaller number of cells engaged in absorbing the acid produced allows for a greater accumulation of acid per cell. Under these conditions, it has been noted that hemolysis is always marked.

THE ROLE OF PEROXIDE AND ACID FORMATION UPON THE COLONY CHARACTERISTICS OF STREPTOCOCCUS VIRIDANS IN BLOOD AGAR

The application of the data at hand results in the following explanation for the appearance of colonies of *Streptococcus mitior* or *viridans* when growing in blood agar plate cultures.

When growing in a medium which allows of vigorous growth in the presence of an abundance of free oxygen, peroxide and acid are formed simultaneously and these substances, diffusing outward from the colonies, cause the green discoloration of the blood corpuscles lying in the immediate vicinity. The cells here are not

hemolyzed by the acid because of the protective action of the peroxide upon them. When the period of active growth of the organism nears its end, or when the growth processes are checked earlier by refrigeration, peroxide formation is diminished or checked altogether, with the consequence that its protective influence upon the blood corpuscles lying beyond the greenish zone is lost and they fall prey to the hemolyzing concentration of acid which continues to diffuse. The multiple zones of green, but intact, and hemolyzed corpuscles described by Brown, are easily explained upon the basis of continuous acid diffusion and intermittent peroxide production, green cells being produced when peroxide is being formed (during incubation) and hemolysis resulting when it was not being formed (during refrigeration).

No attempt has been made to study the chemical changes by which the green color is produced and the cells are protected from hemolysis, but inasmuch as others, Butterfield and Peabody (5), Cole (6), Blake (3), have shown that methemoglobin is formed by an oxidative process by organisms not known as peroxide producers, it seems highly probable that methemoglobin formation is part of the process at least. Methemoglobin, however, is of a brownish cast instead of greenish, hence it appears that the process is not a simple one.

CONCLUSIONS

1. The formation of a green discoloration about the colonies of certain streptococci, and presumably of other green forming bacteria when growing upon blood agar, is due to the action upon the blood corpuscles of peroxide and acid, substances which are produced simultaneously by these organisms when in the period of active multiplication.

2. The hemolysis which occurs about the periphery of the green zone when the period of incubation is prolonged, or when the plate culture is refrigerated while yet in an actively growing condition, is probably due to diffusing acid. Peroxide and acid have an antagonistic action upon blood corpuscles, peroxide tending to discolor and protect them from hemolysis by the acid. Hemolysis results, therefore, only when peroxide production diminishes or ceases.

BIBLIOGRAPHY

1. AVERY AND MORGAN. J. Exp. Med., 1924, *39*, p. 289.
2. AVERY AND NEILL. J. Exp. Med., 1924, *39*, p. 357.
3. BLAKE. J. Exp. Med., 1916, *24*, p. 315.
4. BROWN. Monograph No. 9, Rockefeller Inst. for Med. Research, 1919.
5. BUTTERFIELD AND PEABODY. J. Exp. Med., 1913, *17*, p. 587.
6. COLE. J. Exp. Med., 1914, *20*, p. 363.
7. MCLEOD, GORDON AND PYRAH. J. Path. and Bact., 1923, *26*, p. 127.
8. MANDELBAUM. Zeitschr. Hyg. u. Infektionskrankh., 1907–08, *58*, p. 26.
9. RIEKE. Centrbl. Bakt., Orig. 1904, *36*, p. 321.
10. RUEDIGER. J. Inf. Dis., 1906, *3*, p. 663.
11. SCOTTMÜLLER. Münch. Med. Woch., 1903, *1*, p. 849.
12. SMITH AND BROWN. J. Med. Res., 1915, *31*, p. 455.

LEGENDS.

PLATE I.

Fig. 1. Streptococcus viridans. Deep colony in blood-infusion agar plate which has been incubated for 48 hours at 37°C. The corpuscles lying immediately in contact with the colony are intact but of a greenish color. At the periphery of the zone of green corpuscles, some of the blood cells are hemolyzed, hence the lighter color. x 24.

Fig. 2. Ibid. The same colony which has been refrigerated for 24 hours, following the 48-hour period of incubation. The zone of hemolysis now is very much more distinct. x 24.

Fig. 3. Ibid. Deep colony in dextrose-infusion-blood agar after 48 hours incubation. Growth occurred more rapidly, a greater amount of peroxide was produced and hence the zone of fixed (green) cells is wider than in Fig. 1. x 24.

Fig. 4. Ibid. Deep colony in plain infusion blood agar after five days' incubation at 20°C. At this temperature of incubation peroxide production is slight hence the fixed cells are scarce and hemolysis is prominent. x 24.

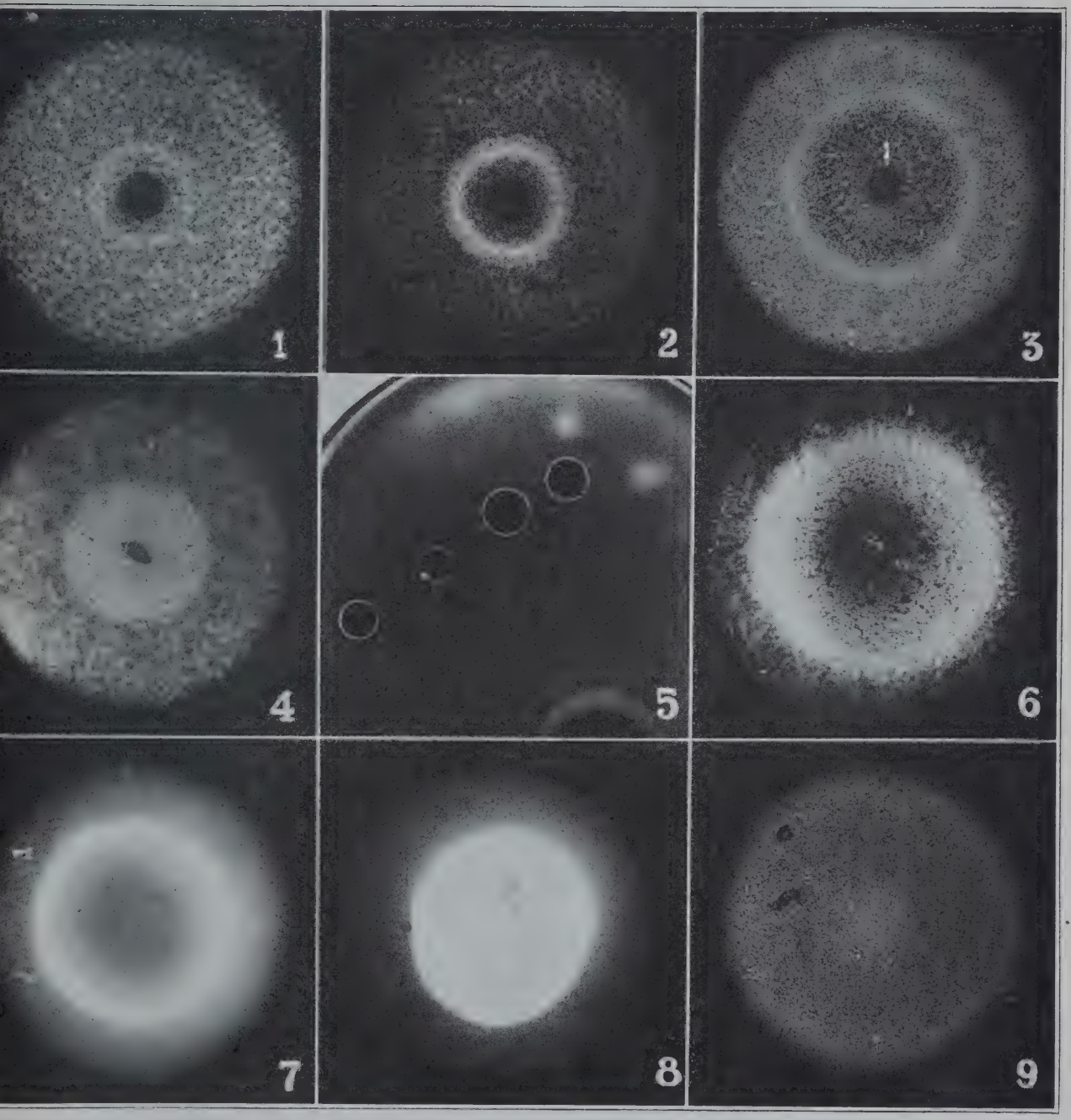
Fig. 5. Effect of drops of concentrated lactic acid upon a blood plate. Photograph taken 24 hours after the application of the acid. The peripheral hemolysis began to appear after several hours and was not complete until the following morning. Natural size.

Fig. 6. Artificial "Colony." Effect of a minute amount of concentrated acid introduced into the layer of medium by a very fine tipped pipette. When not magnified the effect resembled a true colony very closely. (Cf. Fig. 2.) x 24.

Fig. 7. Effect produced by a small drop of 5 per cent lactic acid placed upon the surface of a blood agar plate. Photograph taken 24 hours after the application of the acid. x 24.

Fig. 8. Acid hemolysis produced by placing a very small drop of 3 per cent lactic acid upon the surface of an infusion blood agar plate. Hemolysis is complete. Photograph 24 hours after application of the acid. x 24.

Fig. 9. Same acid solution used for producing effect shown in Fig. 8, after the addition of 1 part of hydrogen peroxide to 5000 of acid, was dropped on the same blood plate at the point shown in this figure. Acid hemolysis has been entirely prevented by the peroxide. x 24.


1
2
3
4
5
6
7
8
9

ARTERIOSCLEROSIS IN DOMESTIC ANIMALS

S. A. GOLDBERG
Department of Pathology and Bacteriology

In the human subject as a result of variations in blood pressure and continuous work, there develops sooner or later a wearing down of the elasticity, changes in the caliber and the strength of the vascular walls. These changes are considered partly as a physiological process and partly as a pathological affection since arteriosclerosis is said to be due to various toxic products.

Arteriosclerosis is described as a fatty degeneration of the intima with proliferation. Calcification or petrifaction of the media may occur secondarily to atheroma or may be primary as is common in old people in the arteries of the lower half of the body. It occurs in the form of rings that have been compared to the trachea of the goose. Individual cases are on record where the media of the entire arterial system was calcified, one in a two days' old child, hence described as congenital.

The typical atheroma is a yellowish white area of fatty degeneration in the intima and spreads as a fatty degeneration into the elastic fibers of the media. This is followed by a regenerative, compensating reaction of the intima so that we get a thickening by proliferating connective tissue.

The etiology is as yet not clearly explained. The mechanical and chemical theory of this affection has been substantiated by the experimental production in rabbits, by adrenalin, hydrastin and numerous other toxic products, of a degeneration of the aorta which may be due to increased pressure, to the effect upon the vessel wall or to both of these effects at the same time. These experimental cases are unlike arteriosclerosis in man, but are necroses of the media with calcification and may be followed by proliferation of the intima. Such changes also occur in man, but do not represent the usual form of this disease. A Russian investigator finally succeeded in producing early changes of arteriosclerosis in rabbits by feeding cholesterin.

These complicated chronic senile changes do not occur in the short lived domestic animals in exactly the same form or with the same frequency. There are, however, cases that are at least similar.

Lyding encountered changes in the aorta in 35 out of a hundred cattle. They consisted of circumscribed thickenings, in flat or slightly concave yellowish white elevations in the intima that histologically showed a hyaline degeneration, fatty and calcified deposits alongside of fibrous proliferations of the intima. He observed similar changes in the horse and in the dog, where these senile changes are mostly to be expected. He found a high grade fatty degeneration of the media with marked deposits of cholesterin, in two cases, in the carotid and in the aorta.

In the etiology he suggests bodily exertions at grazing and draft, as well as toxic influences and nutritive disturbances brought about by intensive breeding and constant wearing in milk production together with poorly ventilated and dark barns.

According to Kitt, dirty white calcified spots in the auricles, as well as in the semilunar valves, are not rare in cattle, particularly in conjunction with tuberculosis. He described a case of irregular masses, in the pockets of the aortic semilunar valves of a horse, that were due to *Strongylus larvae* or to infection. He named this condition *arteriitis fibroplastica petrificans* or *petrificatio atheromatosa verrucosa.* He cites a case of Bollinger of *endoarteriitis villosa verrucosa* at the origin of the aorta next to the semilunar valves and a case of a similar condition in the portal vein, namely *verrucose endophlebitis.*

He claims that the most common condition in the aorta of cattle is the calcified fibrous deposits in the form of round or oblong sharply circumscribed plaques. Because of their resemblance to scales or fins he named this condition *Petrificatio scutellata aortae.* In other cases, besides flattened plaques, there are gravelly, sandy, irregular protuberances, *Petrificatio dispersa aortae.* Tuberculosis petrifaction has been described in tubercular cattle and a tuberculous horse that are said to be an eruption of tubercles into the media under the intima of the arteries.

Kitt also observed small calcified islands in the endocardium and intima of the aorta and pulmonary arteries in swine.

Calcification of the wall of the aorta is a common occurrence in old birds. (Hutyra & Marek, Path. & Therap. of Dis. of Dom. Animals, 1916, Vol. 1, pp. 1185 Trans.)

Lignières describes an enzootic among sheep in Argentine where there was ossification of the lungs with arteriosclerosis. The latter lesion consisted of a marked irregular thickening in the aorta in and near the pockets of the semilunar valves caused apparently by an infection of an intestinal origin.

In nearly 2000 autopsies, including practically all species of domesticated animals, eight cases were encountered in which arteriosclerosis was either directly or indirectly responsible for the death or destruction of the animal. It is quite likely that some cases were missed, particularly where the lesions were minute or in the early stages of the disease. They are presented in order to show that this condition is not so very rare in domestic animals and because they may throw some light on this condition in the human subject.

Case 1. A four year old brown mare, No. 74, weighing about 1200 pounds, that was operated upon for fistulous withers seven days prior to death. She was apparently well up to the time of death. Died suddenly.

Autopsy showed that she was in fair condition as judged by subcutaneous and omental fat. The mesenteric lymph nodes were swollen and congested. The omental lymph nodes in the region of the spleen were hyperplastic. There were several *Filariae equi* in the peritoneal cavity.

The spleen was enormously enlarged, weighing 24 pounds. It was 63 cm. long, 35 cm. wide and 24 cm. thick, and contained scattered nodules ranging in size from 1.5 to 15 cm. in diameter protruding on the surface. On section these were greyish white, cellular, and contained numerous calcified areas. Some of the nodules were surrounded by calcareous material.

Histologically, these nodules were composed of lymphocytes and a few giant cells with necrosis and calcification, apparently *nodular hyperplastic splenitis* with calcification.

The liver showed slight chronic interstitial hepatitis and perihepatitis.

The pancreas was markedly congested and contained an area of hemorrhage 3 cm. x 2 cm. (suggillation).

The kidneys showed an acute parenchymatous nephritis.

The digestive and reproductive systems appeared normal.

Both apical lobes of the lungs showed marked vesicular emphysema.

In the heart there was hypertrophy of the wall of the left ventricle. There were ecchymoses in the endocardium in the apex of the left ventricle. The mitral valves were thickend by connective tissue. The aortic semilunar valves were stenotic due to two areas of calcification about 1 cm. in diameter, partially uniting two cusps. Beginning at the semilunar valves the entire intima of the bulbus aortae and of the aortic arch was corrugated and roughened by fine calcareous points projecting from the surface. The thickening and hardening of the intima extended into the coronary arteries, the common brachio-cephalic trunk, the thoracic aorta and into the left brachial artery. The branches of the brachial arteries were calcified as far as the digital arteries. In the thoracic aorta the calcification extended for a distance of about 30 cm., the amount gradually diminishing until it reached this point. The common carotid and its branches were not affected. In the coeliac artery the wall was thickened. In the intima there were two erosions each about a cm. in diameter and a few very minute calcareous areas the size of a pin point. The intima of the pulmonary arteries of both lungs was hardened by plaques of calcified material resembling the carapace of a turtle. This began about 5 cm. from the pulmonary semilunar valves and extended as far as the terminal arteries. (Figs. 12 and 13.)

All the other arteries were apparently normal. In the brain the meninges were slightly hyperemic. About 100 cc. of colorless liquid escaped from the spinal canal.

In the withers there were fistulous tracts extending into the shoulder. There were numerous calcified areas in the subscapular connective tissue. There was also marked subcutaneous oedema in that region.

In the coxo-femoral joints the synovial membranes were thickened by reddened fibrous tufts. The articular cartilages were smooth and shiny, i. e., normal. The other joints were apparently normal.

No verminous aneurisms were found in the arteries. No strongyli were found either in the arteries or in the intestines.

Case 2. An aged bay mare, No. 145, used for surgical exercises. Previous physical examination showed an increased vesicular murmur at the ventral and diaphragmatic borders with crackling rales. The right chest showed the ribs well sprung with increased resonance. The heart showed a pounding impulse, the second sound being louder and more prolonged. The animal died after administration of a few drops of chloroform.

On autopsy the pericardial sac was distended by about five liters of sero-sanguineous liquid and a large dark clot of blood. The endocardium of the right ventricle was slightly roughened. There was a rupture of the coronary artery with a large hemorrhage in the mass of fat at the base of the heart between the right auricle and the vena cava. The coronary arteries and the aorta at the emergence of the coronary vessels showed the intima roughened, with calcification in the media under the intima. The rest of the heart appeared normal. There were several verminous aneurisms in the mesenteric arteries.

Both kidneys showed nephrolithiasis, the largest calculus being 2 cm. in diameter. The urinary bladder contained a large amount of greenish gritty material with a chronic thickening of its wall.

The liver showed slight cirrhosis with fibrous tufts over the spleen and diaphragm. Adult strongyli were found in the cecum and colon.

Diagnosis — immediate cause of death — rupture of coronary artery.

Fatal illness — arteriosclerosis.

Other lesions — nephrolithiasis.

Case 3. A 22 year old grey mare, No. D 30, become weak and suddenly collapsed. Autopsy showed petechiae throughout most of the organs. The left ventricle of the heart was dilated and the wall thinner than normal. The mitral and tricuspid valves were normal. The aortic semilunar valves were thickened and hardened by roughened calcified nodules.

The aorta was apparently thickened and the intima wrinkled throughout. The extreme end of the aorta and the iliac arteries showed concave, calcified plaques with the lumen widened. (Fig. 6.)

Histologically, the myocardium showed marked degeneration, increase in intermuscular connective tissue with hyaline degeneration of the same. The aorta showed disappearance of the elastic fibres beginning apparently around the vasa vasorum with marked hyaline degeneration in areas and proliferation of connective tissue in the intima and calcification in the media. There appeared to be some fatty degeneration in the intima in places.

Case 4. A cow, No. 110, that had freshened about six months previously. Had been losing flesh since. Showed dysentery with anorexia. Tuberculin test was negative. Destroyed by a blow on the cranium.

Autopsy showed a considerable number of adhesions between the rumen, diaphragm and spleen and numerous reddened fibrous tufts on the omentum and on the peritoneum generally.

The anterior mesenteric and the pulmonary arteries showed calcified areas in the form of irregular plaques similar to Case 1. The calcification was in the media just beneath the intima. In the aorta the intima was extensively thickened and irregular with calcification underneath. This began at a point approximating the free edges of the semilunar valves and extended over an area 9 cm. long covering the entire inner surface of the vessel. Beyond this area there were only individual roughened spots on the intima. (Fig. 1.)

In the heart the endocardium of the right auricle was roughened with areas of subendocardial calcification extending over and almost to the edge of the auricular surface of the tricuspid valves. Aside from this the heart was normal. There was no arteriosclerosis in the coronary vessels.

The mesenteric lymph nodes were swollen and juicy. Two of the bronchial, five of the sternal and the sublumbar lymph nodes were hemorrhagic. The other lymph nodes in the body were normal.

The liver showed some productive hepatitis with areas of necrosis apparently due to *Actinomyces necrophorus.*

The abomasum and the small intestines showed a chronic catarrhal inflammation, being most marked in the ileum. Scrapings from the mucosa showed acid fast bacteria, apparently Johne's disease.

The other organs appeared normal.

Case 5. A four year and six months old Guernsey cow, No. 1344, that died with what was suspected clinically as a traumatic gastritis. She was tuberculin tested at regular intervals with negative results.

Autopsy showed generalized tuberculosis primarily in the uterus, with ascites.

There was hypertrophy of the heart with dilatation of the left ventricle. Beginning at the semilunar valves the aorta contained calcified plaques extending as far as the iliac arteries, apparently not involving any of the branches. In the pulmonary arteries there were areas of calcification at the edges of branching vessels. The left auricle showed irregular raised calcified areas. At the place where there was most calcification the lumen of the aorta was diffusely widened. (Fig. 5.)

Case 6. A sixteen year old Holstein cow, No. 1792 (Glista Ernestine). She was suffering from septic endometritis and destroyed with chloroform.

Autopsy showed some ascites and hydrothorax. There was an old adhesion between the liver and reticulum for an area of 2 cm. in diameter. In the center of this was an abscess 5 mm. in diameter extending into the liver for a distance of 2 cm. The liver around this adhesion showed areas of coagulation ne-

crosis and of fatty degeneration. There was also adhesion between the reticulum and diaphragm. Other old lesions were adhesive pleuritis, mostly on the right side, and some degeneration of the articular cartilage on the femoral heads.

The uterus was not completely involuted, the left cornu being larger than the right. The serous surface was thickened by connective tissue *(chronic productive parametritis)*. The contents of the horns were a cloudy reddish liquid with the cotyledons necrotic and hemorrhagic.

The heart was enormously enlarged, weighing, when empty, 7½ pounds. There was arterial calcification in the form of plaques involving the entire abdominal aorta, extending into the iliac arteries as far as the finer subdivisions. The lumina of the vessels affected were markedly enlarged. (Fig. 7.)

Case 7. A 21 years old Holstein-Jersey cow, No. 1803, used in surgical practicum, and destroyed by chloroform and bleeding from the carotids.

Autopsy showed thickening and roughening of the articular cartilage of the right femoral head. The aorta showed some wrinkling near the base of the heart, with beginning calcified plaques near the coeliac axis. The coeliac arteries were dilated and wrinkled. At the renal arteries and extending to the bifurcation of the iliacs, the aorta showed large calcified yellow plaques some as large as a centimeter in diameter. The arteries branching off at these points were dilated and showed corrugations perpendicular to the long axis of the arteries.

Case 8. A four year old male spaniel, No. 1715, destroyed with strychnine because of hypersecretion of the anal glands. Autopsy showed the heart dilated, the myocardium flabby and somewhat paler than normal. The dilation was mostly in the left ventricle with the wall markedly thinned. In the aorta directly above the semilunar valves there were three calcified plaques. The rest of the aorta and the pulmonary arteries appeared normal. All of the other organs were normal.

Histologically there appeared in Case 1 a proliferation of connective tissue in the intima with hyaline degeneration and deposition of lime salts extending in places deeply into the media with fatty degeneration surrounding the calcified plaques. There was a disintegration of the elastic fibers in and around these areas.

In places the intima showed proliferation, small round cell infiltration with vacuolation. Staining gross pieces of tissue with Herxheimer's fat stain showed the plaques surrounded by a scarlet zone indicating that these vacuoles represent a lipoid substance. Some of these plaques were in the media, the intima in these areas not being affected. (Figs. 14–19.) There were scattered areas of hematogenous pigment in the intima, representing disintegrated red cells caught in the proliferating tissue.

Case 5, Cow 1344, showed the subendocardial tissue to be thickened uniformly throughout the auricle by fibrous tissue containing iregular calcified spots. The myocardium in that zone was prac-

tically all disintegrated, there being a line of muscle fibres just under the endocardium that was still intact, with the cross striations, however, not very evident. The arteries showed changes similar to those of Case 1. (Figs. 9 and 10.)

Case 4, Cow 110, showed thickening of the intima by proliferated fibrous tissue with calcification mostly in the media directly beneath the inner elastic membrane of the intima. (Figs. 2, 3 and 24.)

Case 6, Cow No. 1792, showed a stretching or loss of elasticity of the internal elastic membrane with a disappearance or disintegration of the elastic fibres of the media, leaving an area of hyaline degeneration in the media next to the intima, which became calcified. (Fig. 11). In this case also there was fatty degeneration around the calcified plaques.

In case 8, Dog 1715, the calcified nodules were in the media next to the adventitia in places extending as far as the intima. Around these nodules there was hyaline degeneration. The process in this case seemed to have originated around the vasa vasorum near the adventitia. (Figs. 20 and 21.)

Discussion. In the human subject the etiology generally given is hard work producing increased tension on the vessel wall, intoxication by chemical substances such as alcoholism, and infections, particularly lues. Somewhat similar causes may be ascribed to these cases. In case 1 there was evidence of absorption of toxic products from an infectious focus, namely fistulous withers. The hemorrhages in the endocardium also indicate a toxemia or bacteriemia. In case 2, also, the nephrolithiasis with cirrhosis of the liver indicates possibly some kind of a toxemia. The parasite, *Strongylus vulgaris*, might also have contributed as a cause. Both of these cases had coronary disease which is a common cause of death in human arteriosclerosis.

In case 3 as well as in case 2 there was senility in addition to evidence of intoxication and myocardial degeneration.

In cases 4 and 5 there was probably long continued toxemia, in case 4 from Johne's disease and case 5 from chronic tuberculosis. In cases 6 and 7 there was besides evidence of mild infection, a history of long continued excessive productivity.

Case 6 is that of *Glista Ernestine,* the world record Holstein cow, having produced during her sixteen years of life 202,005.6 lbs. of milk, 7,342.25 lbs. of fat in 12 lactations covering 14 years. She had 13 live, healthy calves with no abortions. A record like this certainly represents an unusual strain upon her vascular system. In addition she had a long standing abscess resulting from *traumatic gastritis.* This possibly had a toxic effect upon the vessel wall.

Case 7 was a family pet for 21 years, also a heavy producer, though a grade cow. Twenty-one years' wear must have had some influence in weakening the aortic wall. Like Glista she had evidence of rheumatoid arthritis and adhesions between the rumen, reticulum and the diaphragm.

There was a remarkable lack of thrombosis in any of these animals. This leads to a belief that the endothelial lining of the intima was not primarily affected. It seems that the elastic fibres were first affected, by a thickening and loss of elasticity, possibly a hyaline degeneration. Later there appeared a deposit of lipoid substance with formation of fatty acids and calcification.

Case 2 in addition to the advanced age, also shows evidence of chronic toxemia as evidenced by cirrhosis of the liver, nephrolithiasis and chronic urocystitis. The strongylosis also was a source of intoxication.

Case 3 shows wear and tear over a period of twenty-two years with the arteries used the most, namely the iliac arteries, affected by calcification.

In case 8 the condition is different than in the other animals. Here the calcification and degeneration are in the media next to the adventitia rather than near the intima. Pappenheimer recently described aortic changes in cases of rheumatism in man. He showed a disappearance of the elastic fibres principally around the vasa vasorum. In this case also there was disappearance of the elastic fibres around the vasa vasorum of the media near the adventitia with the calcification in nodular form in degenerated areas near the adventitia, in one spot extending through the entire media. In syphilitic aortitis there is also an extension of the lesion from the adventitia. In this case the cause may be infection, possibly what would be called *rheumatoid aortitis*. The arteriosclerosis and thromboarteritis so common in equidae resulting from infestation of *Strongylus vulgaris* larvae have purposely been omitted from this discussion since they come into an entirely different category.

SUMMARY

1. Eight cases of varying degrees of atherosclerosis are described.

2. The etiology may to some extent be comparable to that in human arteriosclerosis.

3. The lesions are in many respects similar to those occurring in man.

REFERENCES

SALTYKOW, S. Atherosklerose bei Kaninchen nach wiederholten Staphylokokkeninjektionen. Beitr. f. path. Anat. u. allgem. Path. 1908, xliii, 147.

LYDING, H. Zur Kenntniss der Arteriosklerose bei Haustieren. Zeitschr. f. Tierm. 1907, xi, 359.

LIGNIERES, J. L' Arteriosclerose Epidemique du Mouton. Rev. Gen d. Med. Vet. 1912, xx, 1.

ASCHOFF, L. Pathologische Anatomie, 1923, Sn. 61.

ADAMI, J. G., AND NICHOLLS, A. G. Systemic Pathology, 1911, II, 175.

KITT, TH. Path. Anat. d. Haustiere, 1911, II. 407.

Description of Plates

Fig. 1. Vessels cow 110. About one-half natural size.
A. Aortic semilunar valves (normal).
B. Raised irregular calcified areas on intima of aorta.
C. Similar raised areas on pulmonary artery.
D. Roughened calcified endocardium auricle.

Fig. 2. Aorta cow 110, x 20, showing calcification of intima and of media next to intima.

Fig. 3. Medium sized artery cow 110, x 20, showing thick intima and adventitia with calcification of media. (Note. In all the microscopic figures I is intima: M is media; A is adventitia.)

Fig. 4. Medium sized artery cow 110, x 20, showing calcification and hyaline degeneration of media.

Fig. 5. Aorta cow 1344 about one-half natural size.
A. Near end of abdominal aorta showing scattered concave plaques in areas.
B. Thoracic aorta showing raised calcified spots.
C. Anterior part of abdominal aorta practically covered with various sized concave plaques.

Fig. 6. Iliac artery Horse D 30, showing small concave plaques. (Reduced.)

Fig. 7. Abdominal aorta cow 1792 (Glista Ernestine). (Reduced.)

Fig. 8. Aorta cow 1803. (Reduced.)
A. Widened iliac arteries.
B. Widened coeliac axis. Note the concave plaques scattered through these arteries.

Fig. 9. Aorta, cow 1344, x 20, showing calcification in media just under the intima.

Fig. 10. Intima auricle cow 1344, showing subendocardial calcification and degeneration.
A. Practically normal endocardium.
B. Subendocardial degenerated areas. The black spots in the wide band under endocardium indicate calcification.

Fig. 11. Iliac artery cow 1729, x 20, showing disintegration of elastic fibres. The right half is normal. The light area in the media and intima on left represents degeneration.

Fig. 12. Pulmonary artery horse 74 about one-half natural size. The darker, irregular, somewhat rounded areas represent calcified plaques.

Fig. 13. Aorta and coronary artery horse 74 about one-half natural size.
A. Semilunar valves.
B. Opened coronary artery showing marked calcification.
C. Aorta showing irregular raised calcified masses.
D. Section of aortic wall showing the calcified areas extending into the media.

Fig. 14. Coronary artery horse 74, x 20, showing calcification and proliferation of intima.

Fig. 15. Aorta horse 74, x 20, showing calcification and proliferation of intima.

Fig. 16. Aorta horse 74, x 30, showing calcification and vacuolation of media. The dark oblong area in the center is a calcified plaque surrounded by vacuoles, which proved to be lipoid material.

Fig. 17. Aorta horse 74, x 40, showing vacuolation and proliferation of intima with degeneration of media.
Fat vacuoles in folded and proliferated intima.
Vacuoles in media.
Hyaline degeneration of elastic fibres.

Fig. 18. Taken from left upper corner of Fig. 17 at C, x 100, showing cellular infiltration of intima and degeneration.
Infiltration of small round cells.
Hyaline degeneration of media.

Fig. 19. Another part of aorta horse 74, showing extent of degeneration and proliferative changes.

Fig. 20. Aorta dog 1715, x 20, showing calcified nodules in the media next to adventitia.

Fig. 21. Aorta dog 1715, x 40, showing degeneration around vasa vasorum near adventitia.

A. The larger of the group of vasa vasorum surrounded by a homogeneous degenerated area.

Fig. 22. Aortic semilunar valve. Horse D 30. x 60.

A. Thickened endothelial lining.

B. Hyaline degeneration and calcification of thickened connective tissue of valve.

Fig. 23. Aorta. Horse D 30, x 100, showing two vasa vasorum surrounded by an area where the muscle and elastic fibres are gone, with hyaline degeneration of the connective tissue.

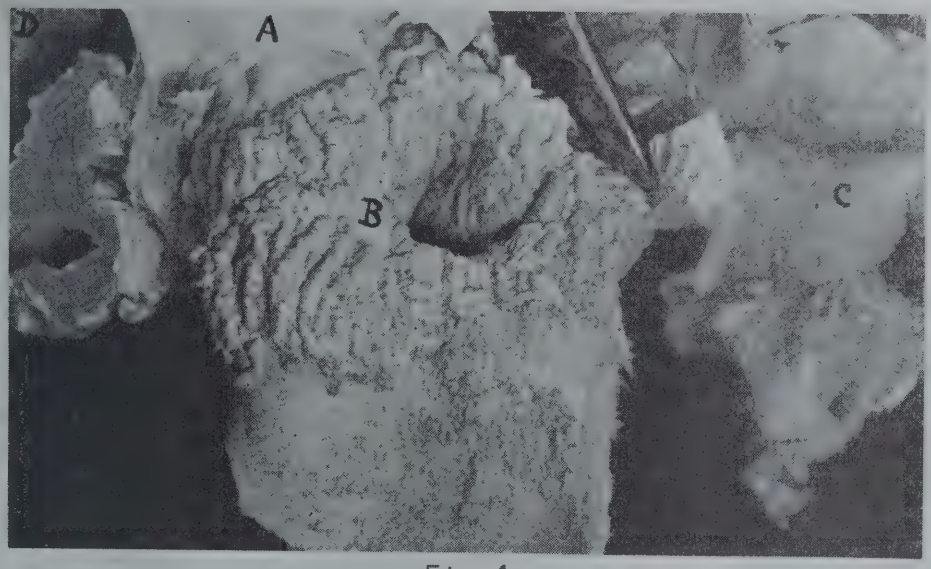


Fig.1

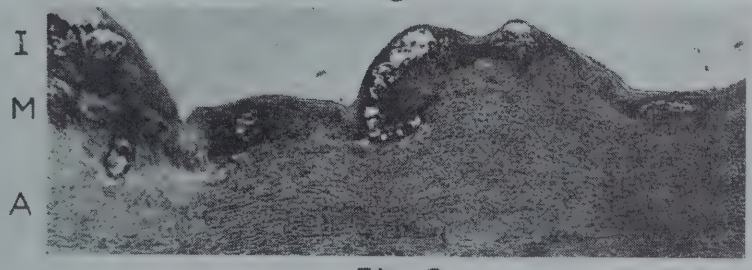


Fig.2

Fig.3

Fig.4

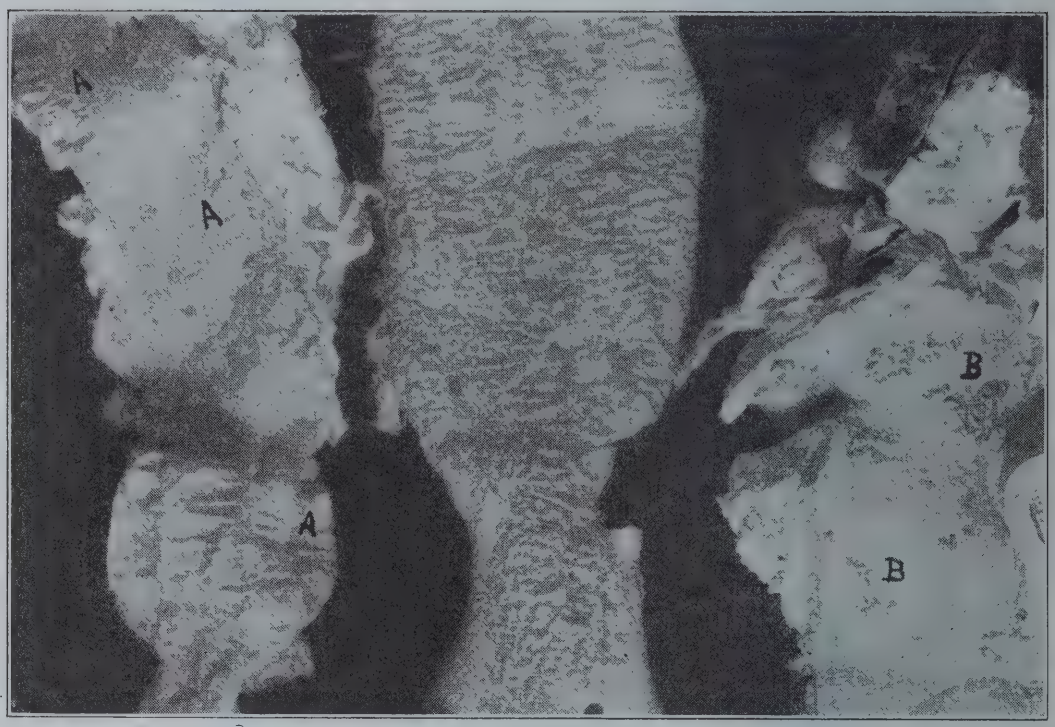


Fig. 5

Fig.6

Fig.7

Fig.8

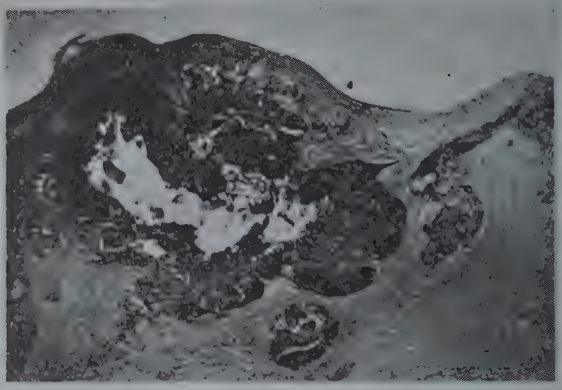


Fig.9

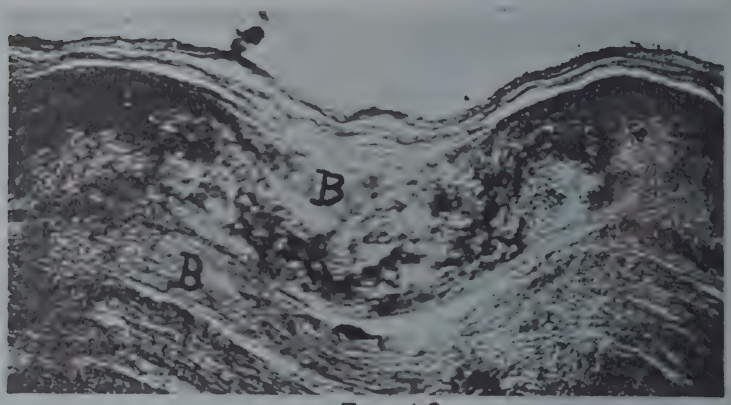


Fig.10

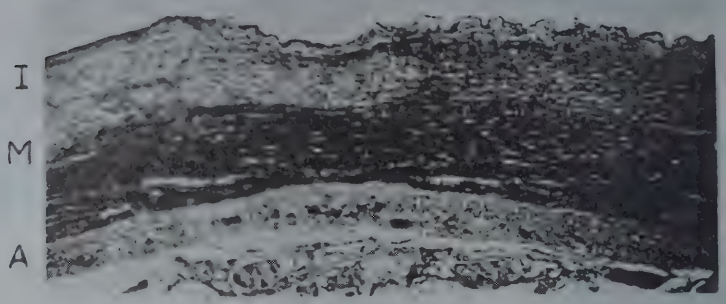


Fig.11

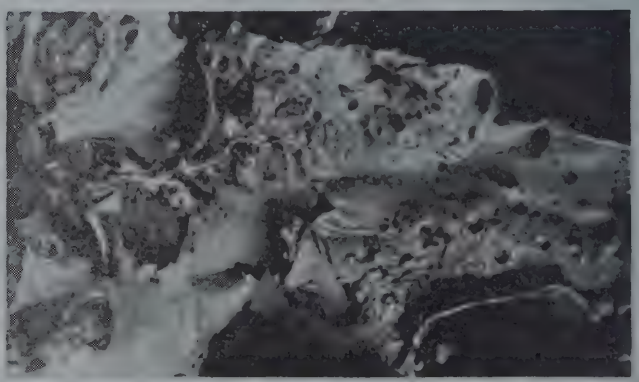


Fig.12

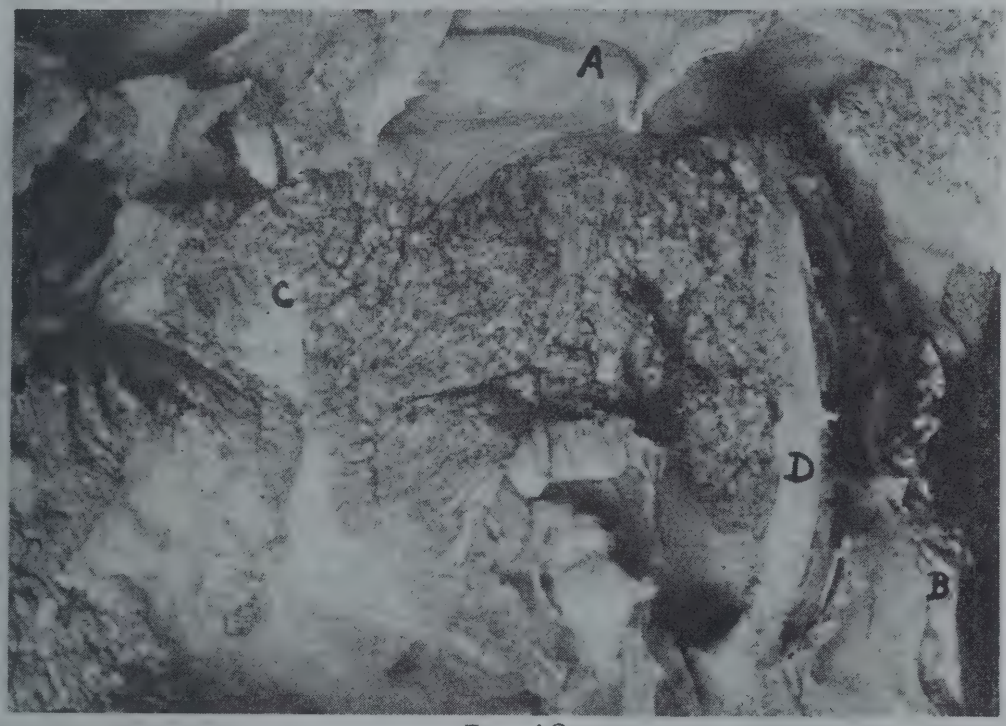


Fig.13

Fig.14

Fig.15

Fig.16

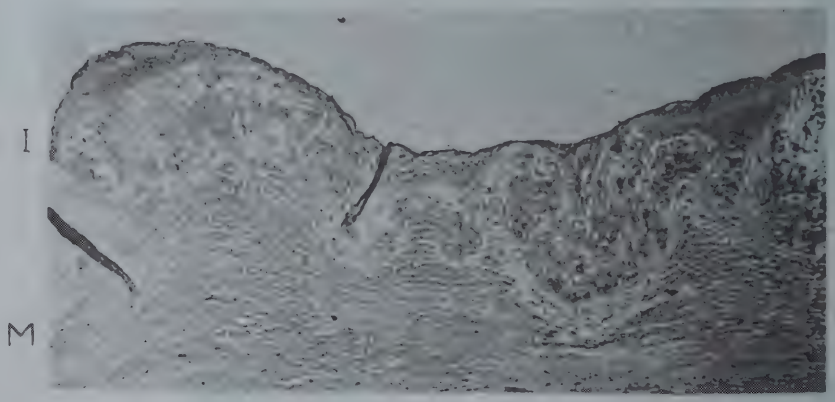


Fig.17

Fig.18

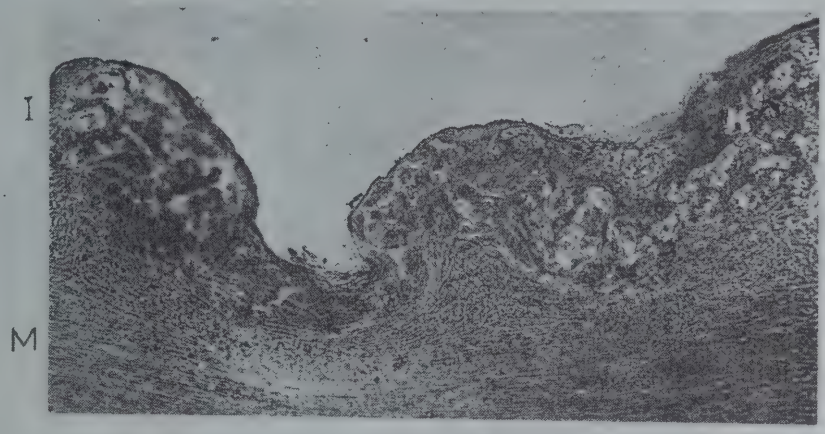


Fig.19

Fig 20

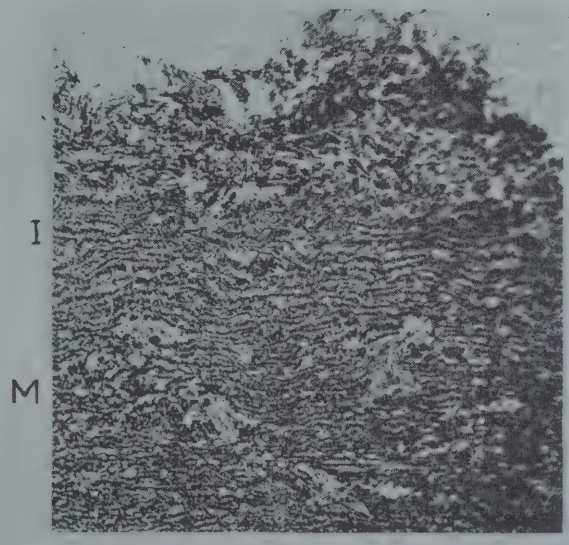


Fig.21

Fig.22

Fig.23

A CLINICAL AND PATHOLOGICAL STUDY OF THE NERVOUS FORM OF CANINE DISTEMPER*

S. A. GOLDBERG AND R. H. VOLGENAU
Department of Comparative Pathology and Bacteriology

Dog distemper is generally known all over the world as the most contagious and destructive disease of dogs. It was first supposed to have occurred in Europe in the second half of the eighteenth century where it was introduced either from Asia or Peru.

Numerous investigators have attempted to separate distemper into various diseases according to the chief symptoms or lesions noted. For example, Trasbot endeavored to divide it into true distemper, la grippe, and infectious pneumonia. Clinically, however, distemper must be regarded as one disease and that disease may be subdivided into four types according to the systems chiefly affected. In the cases studied by us, however, this disease seldom occurred in any one form. It usually showed various combinations. The varieties generally considered are the catarrhal, the exanthematous, the digestive, and the nervous.

The catarrhal form is characterized by an inflammation of the mucous membranes of the respiratory tract. It is the one that Trasbot wished to call the "specific infectious pneumonia." In our cases there was generally a conjunctivitis and rhinitis in conjunction with various other conditions. Superficial and ulcerative keratitis, with the forcing out of Descemet's membrane (keratocele) also occurred. Some cases showed infection of the lacrimal gland, tarsal glands, and lacrimal sac, as well as sudden blindness, without visible macroscopic lesions.

In the respiratory tract, rhinitis is almost universally present, it being of considerable diagnostic significance. Pharyngitis, laryngitis, tracheitis and bronchitis were common, while pneumonia and pleuropneumonia were among the most frequent conditions found.

The exanthematous form was not encountered alone. The eruptions were on the abdomen and inner parts of the thighs, more rarely on the skin of the face, around the eyes, and on the inner surface of the ears. They appeared as small red spots, which soon developed into nodules surrounded by a red zone. Then vesicles formed, which, on becoming infected, developed into pustules. These either ruptured, leaving a raw spot later being covered by a scab, or the pustules did not rupture but dried up, forming a brownish yellow scab.

The digestive form caused considerable trouble by its persistence, by its irregular response to treatment, and the repeated relapses. Some of the conditions observed were: catarrhal, or catarrhal and ulcerative stomatitis, gastritis with frequent persistent vomiting, constipation in the early stages, later profuse watery, fetid, shiny,

* The Cornell Veterinarian, Vol. XV (1925), p. 181.

frothy diarrhea. Frequently the feces were mixed with blood and mucus. In the intestines it varied from a mild catarrh to severe hemorrhagic gastro-enteritis. Icterus was present in a few cases.

The nervous form of the disease will be discussed in greater detail further on in this paper.

The literature on this subject is quite extensive. Most of the work done has been in an effort to determine its etiology.

Jenner (1809) recognized its infectious nature, and that the "miasm" retained its contagious properties for a long time. He was the first to differentiate distemper from rabies, and to recognize the fact that it is not transmissible to man.

The first bacteriological investigations on distemper were those of Semmer (1874), Laosson (1882), and Krajewski (1882), who severally isolated a small bacillus and what was probably a streptococcus from various organs, the blood and urine. Rabe (1883), Mathis (1887), and Marcone and Melone (1888) cultivated streptococci or staphylococci from the pustules, the nasal and conjunctival mucosae, the blood, and various organs, claiming these organisms to be specifically related to distemper. Kitt (1884) questioned the significance of Rabe's finding as he was not able to produce typical distemper by the subcutaneous inoculation of micrococci in young dogs. Jacquot and Legrain (1890) isolated a motile diplococcus from the pustules and found that a distemper-like eruption followed its inoculation, but the lesions were entirely local and did not lead to systemic distemper.

In 1896 and 1908, Galli-Valerio claimed that distemper is due to an ovoid, bipolar, motile bacillus which he recovered from various organs. This organism reacted indifferently to Gram's stain and the cultural characters placed it between the typhoid and septicemia hemorrhagica group. He called it *B. caniculae* and claimed to have produced the disease.

Jess (1899) isolated an ovoid bipolar organism which Galli-Valerio considered identical with his organism. Piorkowski (1905) also found a similar organism, which he believed caused the catarrhal symptoms, while the nervous form he thought was caused by a fine capsuled bacillus.

Von Wunscheim (1905) isolated an organism, which was probably identical with Morgan's bacillus No. 1. Copeman in 1909 claimed to have isolated the cause, a Gram-negative coccobacillus, which he claimed produced distemper-like symptoms and death in one week.

Lignières advanced an organism belonging to the Pasteurella group which he claims is the etiological factor. He was able to recover it only occasionally, and generally only in the early stages from the blood and nasal discharge. He claimed this bacillus was a primary invader, and secondary lesions were due to other organisms. He considered pericarditis as a pathognomonic lesion of the disease.

Phisalix isolated Lignières' organism from septicemia of guinea pigs and claims to have produced distemper symptoms by inoculation into dogs. He failed to find it in fatal cases of distemper. He found chiefly streptococci. Later, instigated by Lignières' findings, he was able to isolate *Past. canis* from blood and viscera of distemper dogs that were killed before secondary symptoms developed.

The specificity of *Past. canis* in regard to distemper has been questioned by Richter and Trasbot, as the former was unable to cure the disease by Phisalix's vaccine.

Carré (1905) claimed that distemper was due primarily to a filterable virus and that the disease as a whole consists of a series of secondary infections by cultivable bacteria. His experiments showed that the nasal discharge at the onset of the disease, when passed through bacteria-proof filters, was very virulent, two drops injected subcutaneously proving fatal to young dogs. The lesions were usually of a septicemic nature, while in more resistant dogs typical distemper symptoms and lesions developed.

In 1906, Lignières confirmed Carré's findings and reversed his opinion of *Pasteurella canis* to the status of a secondary invader. Kragenow and Ferry were unable to confirm Carré's findings.

In 1910 and 1911 Ferry published a detailed account of his researches. He was very careful to prevent accidental contact infection in his inoculation experiments. He claimed that the bacillus which he named *B. bronchi canis* was the cause of distemper, and to quote from his summary: "Koch's law has been fulfilled and cases of distemper have been produced artificially by inoculations with pure cultures, the bacillus being again isolated and grown in pure culture, all under conditions which have precluded infection from other sources."

Independent of Ferry, McGowan isolated this same bacillus from distemper cases. His reports appeared in January and April, 1911.

In 1912 Ferry reported *B. bronchisepticus* as the cause of a severe infection among laboratory animals other than dogs. In January, 1923, Torrey and Rabe published a comprehensive work on canine distemper. Their studies were mostly on the etiology, but some attention was given to symptoms, pathogenesis and anatomical changes. They were able to isolate *B. bronchisepticus* from practically all the parenchymatous organs of the body. Their cultures of the pericardial fluid were negative. They agree with Ferry that the nose is not the primary seat of the multiplication of the cause of the disease, which is contrary to McGowan. They also believe that distemper is caused by a specific agent, but is subject to diverse secondary infections. They claim to have produced an active immunity in susceptible dogs by subcutaneous injections of small doses of this bacillus.

Von Wunscheim (1913) claims that distemper is due neither to *B. bronchisepticus* nor to a filterable virus, but to an organism of the para-typhoid group. He claims to have isolated this organism from all forms of the disease and to have reproduced the disease upon inoculation.

In 1915 Carré discussed the etiology and pointed out the contradictory researches and declared that this obscurity depends upon:

1. Difficulties of research and contradictory results.
2. Impossibility of clinical differentiation between distemper and other diseases of young dogs.
3. Lack of information as to
   (a) Exact lesions and symptoms.
   (b) Virus proper and secondary microbes.
4. Diversity of secondary changes.
5. Multiplicity of microbes encountered in the lesions.

Olsen (1921) isolated a bacillus from five distemper cases which was identical with Pfeiffer's influenza bacillus. Kondo (1923) reported the following results of his researches:

1. Distemper may be transmitted by emulsions of distemper lungs.
2. The filtrates may be injected without apparent consequences.
3. In 85 per cent of the cases, he found *B. bronchisepticus*, 50 per cent in pure culture and 35 per cent associated with streptococci, staphylococci or *Bact. coli.*

Serum of sick animals agglutinated emulsions of *B. bronchisepticus* showing that it is the agent, more or less, that is playing a certain role in the course of the disease. Inoculation of cultures did not always give the same results. Symptoms of an acute septicemia and chronic symptoms resulted. The bacillus isolated was comparable to the *B. bronchisepticus* described by Ferry and McGowan.

Kantrowicz and Lewy (1923) worked particularly on the nervous form of distemper. They worked on 22 dogs. They cultivated numerous organisms, the most prominent being *B. bronchisepticus.* They regard it as a common inhabitant of the air passages of the dog that may cause catarrhal symptoms. They also claim to have observed in sections of the brain a "parasite," a cyst-like structure, composed of a membrane enclosing small granule-like masses in the large nerve cells and in glia cells, resembling "*Eimeria.*"

It is suggested that infection occurs by penetration of this parasite through the capillary walls, which invades first the endothelium, and later the nerve

tissue. They have not proved, however, experimentally, the connection between these bodies and that type of the disease.

The second report of the Distemper Research Council in England (March, 1924) into the cause of distemper and their preparatory work on filterable viruses in general, and investigations into *B. bronchisepticus*, seem to show that the latter organism is not present to any considerable degree on post-mortem, and their results lead them to believe that this organism is a secondary invader.

Babes (1898) working on the effect of various infections upon the spinal nerve cells concludes as follows:

1. Concentration of chromatin in the center or periphery of the cells has little significance in connection with infectious processes of the spinal cord. These forms of chromatolysis may appear as remains of previous diseases, in development of the cell, or from fatigue.

2. It is of greatest significance if following an infection only individual cells or cell groups, the central canal, and vessels are affected finally spreading so that the surrounding cells are involved.

3. The affection of various parts of the spinal cord depends upon the nature of the virus and its infiltration into the cord. For example, *B. pestis* and its toxins infiltrate the nerve cells from the finer vessels of the grey substance and produce a high grade destruction of the nerve cells, a virulent member of the colon group extends into the grey substance from the central canal, the proteins of rabbit septicemia remain localized in the central canal and its neighboring structures, while its toxins injure the neighboring nerve cells. The virus of rabies gets into the cord from the roots, from the central canal, or from certain vessels and produces small foci of perivascular cell proliferation and hemorrhage, and then changes in the nerve cells and their surroundings. In general very virulent bacteria and toxins usually produce less intensive changes than those that affect the spinal cord for a longer period of time.

4. The changes produced are very variable. Lighter acute changes consist of dilation of vessels, lighter cell infiltration, slight edema around the nerve cells, fading or more compact location of the chromatin elements, the appearance of diffuse, finely granular chromatin, as well as chromatin granulations in the nucleus, or in fading of the nuclear substance with a diffuse appearing nuclear membrane. The nucleolus also appears smaller, darker or it may be swollen. It is often displaced.

More marked injury of the cell produces conglomeration, degeneration or disappearance of the chromatin, high grade nuclear and nucleolar changes, desquamation of the peripheral chromatin elements, disappearance of the chromatin spindles in the dendrites, vacuolation in the cells and in the perivascular spaces, with an increased number of cells in and around them, breaking of the appendages of the cell, hyperemia and often hemorrhage in the grey substance, with proliferation and degeneration of small nervous elements and the neuroglia.

High grade injury of the nerve cells is characterized by disappearance of nucleus and nucleolus, invasion of wandering cells into the granular, faint or vacuolated cell itself, the cell appendages having disappeared or being highly swollen, faint, granular or vacuolated. In addition there are found hydropic, hyaline degeneration, pigmentation, segmentation, or peculiar chromatic coagulation necrosis of the cells. All these various conditions are seldom found simultaneously; according to the form of the infection many of these may be found in various groups of cells. Individual cells with all the signs of degeneration may be found in only slightly changed or even normal spinal cord. Bacteria may be found in the nerve cells, usually inside of the vacuoles of the cell protoplasm.

Dexler believes that the fibers are primarily affected followed by parenchymatous destruction, that there is an interstitial cellular exudate that is not confined to definite parts of the brain and cord. He claims that most authors recognize distemper as a specific meningomyelitis.

Bohl claims that there is always developed an infectious myelitis with a chromatolysis of the ganglion cells and even a disappearance of these cells together with all their elements.

Galli-Valerio believes there is a hemorrhagic myelitis.

In 1913, Flexner and Clark described a case of a dog that had paralysis in which they found perivascular lymphocytic infiltration in the cord and in the ganglia simulating, but not identical with, poliomyelitis in man. This case was apparently not distemper as the animal was over three years old. They failed to produce the disease in a monkey and in dogs.

Roman and Lapp, 1925, studied three cases of dog distemper in which they found perivascular lymphocytic infiltration. They also pointed out the resemblance of these lesions to poliomyelitis and to encephalitis lethargica in man.

This work represents a study of 150 cases that came into the college clinic and were diagnosed as suffering from distemper. A careful daily record was kept of the temperature, symptoms, and treatment that each animal received. The animals were often observed 3 to 5 times during the day so that intimate contact with, and a detailed study of, the animals was made possible. Classification of the animals studied into symptomatological groups of the pure form of the disease was practically impossible, as there were many cases with more than one system affected. For this reason the accompanying table shows the disease as it occurred in the pure form and the various combinations of these forms.

TABLE I. SHOWING NUMBER OF CASES THAT FALL INTO EACH TYPE OF DISTEMPER, FOR EACH SCHOOL YEAR 1923–24 AND 1924–25

Type	Catarrhal	Skin	Digestive	Nervous	Catarrhal and skin	Catarrhal Digestive	Catarrhal Nervous	Skin Digestive	Skin catarrhal Nervous	Digestive Nervous	Total cases
1923–1924	26	....	1	6	3	16	27	....	....	6	85
1924–1925	17	....	1	3	2	11	19	....	6	6	65
Total	43	....	2	9	5	27	46	....	6	12	150
Recovered	23	....	1	....	4	12	1	....	....	....	41
Improved	....	....	....	1	....	....	10	....	....	1	12
Died or destroyed	20	....	1	8	1	15	35	....	6	11	97

The catarrhal cases are not very widely separated in numbers and would perhaps be nearly equal if an equal number of yearly cases were studied. The same applies to most of the other types. The pure skin type was not encountered.

No pure cases of skin and nervous form of the disease uncomplicated by catarrhal symptoms also, were encountered, so in this one type we have the triple form of the disease. It is interesting to note that no cases of this type were encountered in 1923-24, while up to the present time we have six cases this year.

Table II shows some of the other conditions that were associated

with the disease. The relative frequency can be determined when the number of cases for each year is noted.

Abscesses were more common in the first year than the second. This perhaps may be attributed to the larger number of hypodermic injections given. Every antiseptic precaution, however, was used to prevent this. Some of these may have been due to contusions, acquired while the animals were in violent convulsions or in their aimless wandering and running into the hard walls of the kennels.

Stomatitis was much more frequent the first year than the second. One in particular in a Springer spaniel was of the ulcerative type. These ulcers spread rapidly and penetrated deeply. They responded quickly to painting with 10 per cent silver nitrate, followed by rinsing the mouth with antiseptic solutions.

Infection of the lacrimal gland and sac occurred in a few cases. From these were isolated *Staphylococcus albus* or *Streptococcus pyogenes,* or both. They readily responded to treatment.

Among the diseases of the membrana nictitans we encountered one patient in which the membrane was quite large and rolled up, outward and medially. These membranes were so prominent that they tended to keep the eyelids somewhat separated near the medial commissures, and a persistent discharge was present. Treatment consisted of the removal of the membranes, and the use of ointment. Here we might say that diseases of the eye may give pseudo-distemper conjunctivitis symptoms, but, of course, other distemper signs would be absent. Distemper cannot be diagnosed by conjunctivitis alone.

Hypopyon, or collection of pus in the anterior chamber of the eye, occurred in two cases, one of these animals having recovered. Treatment consisted of puncturing the cornea and aspiration of the pus. The dog made an uneventful recovery. Recent observation shows normal sight, the only evidence of previous injury being a slight whitish connective tissue scar about 1 mm. in diameter at the corneal limbus, apparently not interfering with vision.

Simple and ulcerative keratitis were prominent as rather troublesome, apparently painful and devitalizing influences to the dogs during the course of the disease. The ulcers for the most part were checked in their early stages by the use of strong phenol solution on the anesthetized eye, after first outlining the ulcer with fluorescin solution. Subsequent treatment consisted of 5 per cent yellow oxide of mercury ointment massage. In two cases, however, both corneas in both animals were almost totally destroyed. One of these animals was destroyed, when partly recovered, because of this condition; the other died shortly after being entered in the clinic. The majority of cases healed promptly, the only trace remaining being a whitish connective tissue scar. Several cases, however, were complicated by blindness and other nervous disturbances. One dog that partially recovered remained blind in one eye and partially blind in the other.

These other lesions encountered, though not in themselves sufficient to cause death, were devitalizing to the animals and helped to lower their resistance to other secondary infections. They are therefore considered worth mentioning.

TABLE II. OTHER LESIONS THAT WERE ASSOCIATED WITH DISTEMPER

LESIONS	Abscesses	Stomatitis	Infection of lacrimal gland	Infection of lacrimal sac	Disease of membrana nictitans	Hypopyon	Keratitis and ulcerative keratitis
1923–1924...	4	3	2	1	2	1	8
1924–1925...	1	1	3	1	.....	1	6
Totals....	5	4	5	2	2	2	14

Infinitely more important were conditions that undoubtedly influenced the course of the disease to a marked extent. We might term these the parasitic and nutritional disturbances that altered the course of the disease.

Feces examinations were made on nearly all of the animals. Many of them were made on the day they were entered in the clinics. The method of examination was usually direct, i. e., a smear made on slide, a small amount of 10 per cent caustic potash added and smears examined for parasite eggs. In other cases centrifuge methods were used. Undoubtedly many cases that were infested with internal parasites were missed by this method, due to lack of the presence of eggs in sample examined, or to the failure of eggs to be in the feces at the time of examination. Tapeworms were discovered usually by finding the segments in the feces or around the anus and tail when taking temperatures. In relatively rare instances *Taenia pisiformis* eggs were found in the feces smear, due undoubtedly to the bursting of one or more segments in the intestinal tract.

In this manner 76 of the animals examined were found to be infested with from one to three different types of internal parasites.

Animals affected with ankylostomiasis seemed to stand less chance against the ravages of this disease than animals infested with other parasites.

Eighteen of these cases were complicated by rickets. This nutritional disease was indeed an important factor in determining the course of the disease. Animals affected with rickets plus one or two types of parasites and in a grossly unthrifty condition were hopeless from the start. Much study is needed on proper feeding and nutrition of small animals. Dogs of the German shepherd breed were most frequently affected. This breed in general seems to be the most susceptible to all forms of dog diseases, bacterial, nutritional, parasitic, and others.

Follicular mange was encountered in nine of the cases. There seemed to be no response of this disease to treatment, while the animal suffered from distemper, probably due to the more favorable conditions that were present for parasitic growth. It is interesting to note here that in one case after apparent recovery from distemper, the hair fell out in circumscribed patches, chiefly on the sides and back, the skin appeared perfectly healthy and microscopic examinations were negative. After a time the hair grew in. It was possibly a case of the so-called alopecia in mild form.

Table I also shows the number of animals having each type of the disease, that recovered, partially recovered, or died. The greatest number of recoveries occurred in the catarrhal form, and this was around 50 per cent. Only one case that had nervous symptoms appeared to have completely recovered. This case was in a Springer spaniel that had choreic twitching of muscles around one eye. This disappeared after about five weeks.

The partially recovered were all nervous cases. They consisted of from partial to total posterior paralysis, or chorea of various muscles of the body. The animals that were deemed hopeless were destroyed. Pneumonia was the most common terminal lesion.

In the study of these 150 cases, emphasis was placed upon the nervous form of the disease. Each nervous case was studied as to symptoms, course and treatment. Each case was allowed to live until death was imminent. Every effort was made to remove the nervous tissue as soon as possible after death. Animals that could not live from one evening until the following morning were destroyed to avoid error when it came time to study the tissues of these animals. When possible, the brain and spinal cords were removed within one hour after death and placed immediately in 10 per cent formalin solution. The cord was cut into suitable pieces for quick fixation and to prevent bending during fixation. After fixation in formalin sections, were run through by the Marchi and Nissl's methods, as well as by hematoxylin and eosin, toluidine blue, and Giemsa stains.

In general the animals showed an intermittent type of fever. The temperatures when the animals were entered in the clinic were in practically all of the nervous cases relatively high. In most of these cases the temperature was higher on the next day, then usually dropped on the third or fourth day. It was, however, extremely variable and not much emphasis can be placed upon it. For the most part the temperatures ran between 102° and 104°F. During convulsions the temperature rose usually around 103.4° to 104° F. It was noted, however, that when paralysis came on suddenly there was a rise of temperature, 105°–106° F., before the onset of paralysis. The temperature would be running along between 102° and 103° F., then this sudden elevation and the next day paralysis. In one case where on post-mortem the animal showed extensive petechial hemorrhages throughout the brain substance on macroscopic examination, the temperature went up to 107.6°F.

When the animals became comatose the temperature was invariably subnormal. One case, however, where the animal was conscious, with excitement and howling continuously day and night, the temperature was subnormal for a week. The animals lived from 24 hours to a week with subnormal temperatures, with invariably fatal termination. (Temperatures below 99.5°F., are considered subnormal.)

The symptoms shown by the animals suffering from the nervous form of the disease have been classified under four headings (after Udall) namely:

1. Disturbance of consciousness
2. Motor irritation
3. Derangement of senses
4. Disturbances of motility proper.

The total number of animals showing nervous symptoms of any sort was 74. The two years were about equally balanced. The same animal may show more than one variety of symptoms. All animals that showed various degrees of depression were not in that condition continuously, some giving way to fits of excitement and convulsions.

Disturbances of consciousness in the form of depression or dullness were present in practically all of the animals studied, including those that suffered from forms of distemper other than the nervous type.

By sopor is meant a semi-comatose condition, although the reflexes are still present. In coma, however, unconsciousness and suppression of reflexes are complete.

Under excitement were grouped only those animals that gave way to long continued howling spells lasting for hours, and that were stopped only by putting the animals under strong sedatives, such as chloral, sodium barbitol, and morphine.

Under delirium are grouped those animals which, almost without exception, after convulsions would attack anything within reach and were unsafe to handle in any manner. Under hallucinations were grouped three animals that showed symptoms that could not be classified in any other way, such as where the animals appeared to perceive something that was not present, biting at imaginary objects, etc.

In all there were 71 animals that showed various psychic derangements. They were not necessarily all different animals.

Perhaps the chief nervous disturbance that occurs in distemper comes under the heading of motor irritability. These symptoms include anything from mild twitchings of individual muscles to convulsions. Most of these symptoms are self-explanatory. Clockhand movements, observed in three cases, consisted in pivoting or circling either to right or left with one hind leg as the stationary axis around which the body rotates.

Rotation in a transverse axis is much the same as falling over backward. One animal of this type was observed to stand up against the side of the kennel on his hind legs, then suddenly go

over backwards. He would also rotate over backwards starting from a standing position.

Rotation on a longitudinal axis was noted in one case. This animal when placed on the floor would roll over and over until she came up against a wall. After this symptom came on she never got upon her feet again. For the most part she lay on her back or partly on her side and partly on her back against one side of the kennel.

Chorea or clonic spasms of muscles was of very frequent occurrence. This condition may affect any group of muscles, and it would be really interesting for some one to work out the relative frequency with which certain muscle groups were affected. The tail was seldom involved, the temporal and masseter muscles perhaps most frequently, leg muscles occasionally, and more rarely other muscles.

Tonic spasms or trismus affected the masseter and other jaw muscles, resulting in a firm locking of the jaws, which resisted the most powerful efforts to open them.

Convulsions were the most prominent individual symptoms, being shown by 42 animals. Often it was the first symptom noted. They started with champing of the jaws, salivation and clonic spasms of the muscles of the face and head, becoming more violent, finally spreading to other muscles and muscle groups until the musculature of the entire body was involved, the animal going down, thrashing violently, sphincters relaxed, feces and urine voided, foamy salivation frequently flecked with blood from injuries to the tongue and lips. The spasms subsided to a considerable extent and the animal lay exhausted for a time with occasional clonic spasm of muscles or muscle groups. Finally they staggered to a sitting or standing position. Some were plainly uncertain as to where they were, would stagger, bump into objects, fall, rise again, or lie down and rest.

Excitement of any form seemed to be sufficient to precipitate the animal into convulsions. Removal of the animal from the kennel for treatment, pilling, slamming the kennel door, sudden barking of other animals, dropping of some utensil, were sufficient, although they also occurred when absolute quiet prevailed. Sometimes the convulsions were intermittent, occurring at intervals of a week or more; at other times the animal went from one convulsion into another, finally terminating in death.

Paddling, or walking movements made by the legs, while the animal was in a semi-comatose condition, and lying on its side, was encountered in six cases. This was usually accompanied by some excitement.

Clonic spasms, or champing of the lower jaw, present at all times, and not associated with convulsions, was noted in ten cases. This symptom, however, was also prominent in all convulsions.

Opisthotonus and pleurothotonus occurred in two and three cases respectively. In all, 120 examples of different forms of motor irritation were noted.

Derangement of senses was not of such great importance outside of derangement of sight. Paresthesia was noted in one case. This animal, an English shepherd, had in all probability an intense itching on the inside of the right thigh. He chewed at it continuously until the entire inside and over the front part of the thigh and stifle joint were entirely denuded of skin. The removed skin was devoured by the animal. During this process the animal did not appear to suffer any pain. No attempt at treatment was made due to viciousness. He lay around for some weeks, finally dying in violent convulsions, with copious hemorrhage from the mouth.

Derangement of sight occurred in the form of so-called amaurosis, that is, no visible macroscopical lesion was observed. This type of blindness may end in sudden recovery, partial blindness, or total permanent blindness in one or both eyes. The pupil was invariably widely dilated and did not readily respond to myotics.

Hypoesthesia or decreased sensibility occurred in practically all paralytic cases. Sensibility was only decreased. Paralyzed animals appeared to suffer little pain from extensive decubital sores, insisting on lying on the sore side, even rolling back on that side when otherwise placed. Stimulation, chiefly rubbing, seemed to be appreciated. Complete sensory paralysis along with the motor did not exist.

Under disturbances of motility proper we have complete motor paralysis (paresis), all four legs being paralyzed, and paraplegia or the so-called posterior paralysis, so frequently seen in distemper. This paralysis, however, did not involve the tail. Of hemiplegia, or paralysis of one leg, we had three cases. One went on to paresis, another was discharged and got along well on three legs, the third went on to paraplegia. Partial paraplegia was frequent. It consisted of partial loss of control of hind parts, stiffness of gait, unsteadiness, slowness in motor response, short steps, etc. Some of these went on to complete paraplegia, others recovered to some extent, while many were discharged in that condition and no further record was obtainable.

Seven blood examinations were made on distemper cases. These animals were of various ages and stages of the disease. They showed a marked polymorphonucleosis. One showed an eosinophilia with absence of intestinal parasitism. Numerous vacuolated polymorphs were encountered in these cases. One animal that had recovered from the disease, but returned later because paralysis developed, showed a marked anemia, with the presence of immature forms of red cells in the circulating blood. Another animal suffering from anemia finally recovered.

Bacteriological study was made on twenty cases. When an animal was entered in the clinic cultures were taken from the nose and eyes. If other secondary infections occurred during life these were cultured. On death of the animal various other organs were cultured. The spleen and kidney very rarely showed bacterial growth on culture media.

From the purulent exudate of the eye chiefly staphylococci, streptococci and *Pseudomonas pyocyaneus* were recovered. *B. bronchisepticus* was isolated in two cases, both in the early stages of the disease. Similar organisms were obtained from the nasal discharge. *Bacterium coli* was continually present in many of the cultures. It seems to spread all over the body in comatose animals that linger on for some time. In animals that succumbed to the disease, streptococci were isolated from the liver in two cases, staphylococci in two cases and colon organisms in seven cases. In the pneumonic lungs cultured staphylococci and *Bact. coli* were the chief organisms found, with *B. bronchisepticus* in three cases. The heart's blood was for the most part negative. Streptococci were isolated in two cases, *Bacterium coli* in one. From the trachea, streptococci were obtained in four cases, staphylococci in one case, *Pseudomonas pyocyaneous* in two cases. From the bronchi in only one case was *B. bronchisepticus* obtained. Staphylococci were chiefly the organisms isolated, streptococci more rarely, and *Bact. coli* were present in most of the cases. In the cerebrospinal fluid two cases showed diplococci and *Bact. coli* and one case gave *Bact. coli* in pure culture. The other cases cultured showed no growth. From infections of lacrimal sac and gland, streptococci and staphylococci were the organisms isolated. In one case of pleuritis, *B. bronchisepticus* was obtained in pure culture.

The results of treatment were as follows: From distemper serums and bacterins of various manufactures we were unable to derive any benefit. Other sera were tried without any satisfactory results. All these animals received triple arsenates, with strychnine content according to the size of the dog, never over 1/120 grain, creosote 1 minim, and 1 dram of cod liver oil twice daily.

Hypodermic administration of distemper bacterins and a preparation of sodium morrhuate recommended by some, were also tried. Intramuscular injections of metaphen—a mercurial with high bactericidal action—in high dilution, were used. Some recovered, some died, but the percentage remained the same as those that did not receive these experimental injections.

Autogenous bacterins were used on several animals in increasing doses. In the cases used, some benefit seemed to be derived from their use. They were used only in cases of chronic nasal or eye discharge which persisted after apparent recovery.

Relatively large doses of quinine sulphate (10 grains per day for three days, then reduction of dosage to two grains per day) on catarrhal cases in early stages seemed to be useful, as all cases receiving this treatment thus far have recovered. In general our treatment has been purely symptomatic.

For the eye discharge no ointment seemed to work better than 1 per cent copper sulphate in lanolin and white petrolatum. This ointment would stop the discharge where all others failed. In only one case has it failed, and this where *Pseudomonas pyocyaneus* was obtained in pure culture. That case remained with a chronic unilateral eye discharge.

For the nose, with excessive discharge, sore cracked nostrils, etc., we used an ointment of 2 per cent menthol, eucalyptus and camphor in white petrolatum. The ointment seemed to relieve breathing, lessened the discharge and prevented erosion of the nose and skin.

In pneumonias, atropine sulphate 1/100 grain per os seemed to give some relief, especially if the breathing was very difficult. Stimulants, expectorants, liniments to the chest wall, etc., were also used.

In the digestive form, for stomatitis, washing of the mouth with a mild antiseptic and touching up of ulcers with 10 per cent silver nitrate; for gastritis, usually in the milder cases, gastritis mixture (bismuth subcarbonate, soda bicarbonate, phenol and water) was given. With very persistent vomiting, cocaine solution containing one grain to an ounce of water was given.

Treatment for parasites was not generally applied if the animal was in such condition that certain death would result. Otherwise they were treated according to the parasite found.

Severe diarrhea was hard to check. If the animal was strong enough, it was first cleaned out with mineral oil and castor oil, equal parts, followed by astringents, protectants, carminatives and sedatives. Those chiefly used were alum, tannic acid, bismuth, tincture of opium, sulphocarbolate and various other proprietary antidiarrheal remedies.

Skin eruptions were so benign that as a general rule no treatment was needed, or the usual opening and disinfection of the pustules followed by a dusting powder.

Little could be done for those animals suffering from the nervous form. Many drugs were used, with little or no result. As a general rule, if the feces were negative and no evidence of parasites in the ears, the animals were given sedatives. They invariably went to pieces, however, and had to be destroyed, or they died. Only two sedatives have given us uniformly good results—chloral hydrate, and elixir sodium barbitol.

As to diet, the chief thing is to keep the animal eating and feed easily digested, nutrient foods. Keep coaxing them along on tempting foods until they will eat. Forced feeding is necessary at times, especially if the animal is too weak to take food.

Cleanliness, fresh air, sunlight, quiet and rest are the chief essentials, and the avoidance of draughts and sudden changes of temperature.

The pathological changes are mostly microscopic. Macroscopically the only changes encountered were some congestion of the meninges, with occasionally areas of hemorrhage in the meninges and linings of the lateral ventricles. On section the brain stem appeared markedly congested and in some cases the lateral ventricles were distended, occasionally to such a degree that the cerebral cortex was atrophied (Fig. 2). In two cases there were reddened areas of softening about a millimeter in diameter, extending from the decussation through the pons.

From the symptoms it would appear that the lesions were located largely in the cerebellum and the vestibular area. Histologically, the most marked change was in the region of the brachium pontis and the pyramidal area. Recently an investigator found that experimental injury to the pyramid produced trismus of the masseter muscle. In these cases champing of the jaw with trismus was present in practically all of the animals showing nervous symptoms of brain origin. In the cord the lesions were mostly in the white matter and not confined to any one tract, but the lateral cerebello-spinal tract appeared to be more frequently involved.

In the cases that showed convulsion from the start and lived only a short time, the principal change found was hemorrhage in the brain substance as well as in the perivascular spaces (Fig. 3). This condition would appear as hyperemia with the unaided eye. In cases of longer duration, Marchi method showed myelinic degeneration as well as hyperemia and hemorrhage (Figs. 6 and 7).

In cases that persisted for a longer time there was vacuolation where the nerve fibers were entirely gone. Between these vacuoles there was hemorrhage or small round cell infiltration (Figs. 9 and 14). This, of course, is a form of softening. In a large number of cases, in addition to the above changes there was perivascular infiltration of small round cells (Fig. 8). These changes were encountered in the brain where the animals showed various brain symptoms and in the cord where the animals showed paralysis without any psychic symptoms.

These lesions were encountered previously during a study of distemper and presented before the American Association of Pathologists and Bacteriologists, May 4, 1922.

At that time it was thought that some of the changes, particularly hemorrhage and hyperemia as well as vacuolation, might have been post-mortem. In this study the central nervous system was obtained within an hour after death and a number of the animals were destroyed by bleeding from the carotids. In these animals we also found the hyperemia and the hemorrhage as in the previous study. Emphasis was placed on the purely nervous form because it was previously noted that animals suffering from parasitism may show nervous symptoms and even brain lesions similar to those found in distemper. It is for this reason that parasitism and distemper are often confused. Such was found to be the case in foxes as well as in dogs.

Some of the symptoms closely resemble those of rabies. Occasionally brains of distemper dogs are sent in for diagnosis as being suspicious of rabies. In some of these animals, the symptoms so closely resembled rabies that we were obliged to examine them for the presence of negri bodies. These were not found.

The changes in the cells we have encountered, consisted in the disintegration and finally disappearance of the tigroid substance or Nissl's granules (Fig. 13). The Purkinje cells in the cortex of the cerebellum also showed vacuolation (Figs. 10 and 11). These changes in the cells are not specific as they are found to a lesser

degree in fatigue as well as in a number of other conditions. That these changes do not necessarily involve a loss of the cellular elements is evidenced by the fact that one of our cases that suffered from posterior paralysis recovered the use of the hind parts in about two years.

The perivascular infiltrations would cause a disturbance of the circulation of cerebrospinal fluid, and under certain conditions produce a distension of the central canal or of the lateral ventricles. In general, in its effect upon the tissues, the virus seems to resemble that of rabies, if the assertion of Babes is found to be correct, that is, in the presence of hemorrhage and perivascular infiltration. In this disease there is frequently found a distension of the central canal with albuminous material (Figs. 4 and 12). This would probably indicate that the cerebrospinal fluid is rich in albumin. It seemed possible that the virus might enter the central nervous system from the cribriform plate. In that case we should expect to find marked changes in the olfactory lobes. We were unable to find changes in this part of the brain. It must enter in some other manner, since many animals show posterior paralysis only without any evidence of affection of the brain. It is quite possible that the virus enters by way of the capillary endothelium as suggested by Kantrowicz and Lewy. Their drawings of motor cells showing what they claim as possible parasites appear to be, in our opinion, a further stage of chromatolysis. Their cases seem to be of a lower grade of infection than was the case in those encountered by us. We have observed similar changes in a case of focal tuberculosis in a human brain.

The lesions encountered in these cases of canine distemper do not resemble those of encephalitis lethargica in man since in the latter the pathognomonic lesion is a diffuse reddening of the brain with numerous hemorrhages, macroscopically visible, and in some cases thrombosis and infarction. The lesions resemble more closely acute poliomyelitis in man, but even here there are distinct differences. We have not encountered infiltration of polymorphonuclear leucocytes in the perivascular sheaths or in the nerve tissue in any of our cases. Areas of softening were not so common, and no "granular corpuscles" were encountered. The comparisons were made with the descriptions of E. F. Buzzard and J. G. Greenfield's Pathology of the Nervous System, and with slides obtained from Dr. Roman of Buffalo.

From a study of these 150 cases the following conclusions seem to be justified:

1. That where the brain is involved the symptoms are referable to lesions in the cerebellum and the pontal region.

2. That the lesions are hyperemia, perivascular and other hemorrhage, perivascular round cell infiltration, chromatolysis of the nerve cells and vacuolation of the white matter.

3. That the ascending tracts are occasionally affected but the descending tracts are more frequently injured.

4. That in the catarrhal form quinine seems to give better promise than other remedial agents.

Acknowledgment. Dr. H. J. Milks offered many helpful suggestions during the course of this work. W. J. Gibbons assisted in the bacteriological and hematological work. R. V. Gibbons assisted in histological technique.

REFERENCES

BABES. Berlin. Klin. Woch., 1898, xxxv, 6, 36, 56.
CARRÉ. Rec. de Med. Vet., 1906, lxxxiii, 389.
COPMAN. Cited by McGowan, Proc. Roy. Soc., London, 1900, 67, 459.
DEXLER. Ergeb. d. allgem. Path., 1900–1, vii, 401 (Lit.).
FERRY. Jour. Inf. Diseases, 1911, viii, 399, and Jour. Path. and Bact. 1915, xix, 488.
FLEXNER AND CLARK. Jour. Exper. Med., 1913, xvii, 577.
GALLI-VALERIO. Centralbl. f. Bakt., 1896, xix, 694, and 1908, xli, 563.
JACQUOT AND LEGRAIN. Rec. Soc. Med. Vet., 1890, vii, 149.
JESS. Centralbl. f. Bakt., 1899, xxv, 541.
KITT. Ref. Kolle und Wasserman, 1903, ii.
KONDO. Rec. de Soc. Med. Vet., Mar. 15, 1924, 175.
KRAJEWSKI. Rev. d. Tierheilk. u. Viehz., 1881, iv, and 1882. Cited by Richter.
LAOSSON. Inaug. Diss. Dorpat, 1882. Cited by Richter.
LIGNIÈRES. Bull. Soc. de Med. Vet., 1906, lx, 622.
M'GOWAN. Jour. Path. and Bact., 1911, xv, 372, and xvi, 257, and Vet. Jour., London, 1912, lxviii, 7.
MAJA. J. A. V. M. A., 1913, xliii, 650.
MARCONE AND MELONE. Giorn. dell' anat. e della fisiol. 1888. Cited by Galli-Valerio.
MATHIS. Rec. Soc. de Med. Vet., 1887, iv, 229.
OLSEN. Ref. Jour. Vet. Med., 1922, xvii, 233.
PHISALIX. Revue Veterin., 1901, lviii, 538.
PIORKOWSKI. Berlin tierarztl. Wochenschr., 1905, 830.
RABE. Wochenschr. f. Tierheilk. und Viehz., 1883, xxi, 126. Cited by Richter.
RICHTER. Die Hundestaupe. Dessau, 1908.
ROMAN AND LAPP. J. A. V. M. A., 1925, lxvi, 612.
SEMMER. Deut. Zeits. f. Tiermed. u. vergleich. Pathol., 1875, i, 204. Cited by Richter.
TORREY AND RABE. Jour. Med. Research, 1912–13, xxii, 291 and Jour. Comp. Path. and Thera., 1913, 69.
TRASBOT. Rec. de Med. Vet., 1903, lxxx, 343, 391.
UDALL. Cornell Vet., 1922, xii No. 2, 101.
VON WUNSCHEIM. Archiv. f. Hyg., 1905, liii, 1 and Vet. Record, 1915–16, xxviii, 313.

DESCRIPTION OF PLATES

PLATE I

Fig. 1. Section of brain dog 1526. Natural size.

A, Cerebral cortex slightly atrophied; B, Distended lateral ventricle; C, Slightly congested brain stem region of pons; D, Hypocampus.

Fig. 2. Section of brain dog 1521. Natural size.

A, Marked atrophy cerebral cortex; B, Marked distension lateral ventricle; C, Hemorrhage and marked hyperemia, cerebral piaarachnoid; D, Brain stem fairly congested; E, Section of hypocampus showing congestion.

Fig. 3. Pyramid brain dog 1624. x 150.

Showing three vessels with hemorrhage into the perivascular sheath.

Fig. 4. Distended central canal cord of dog 1527. x 50.

A, Coagulum in the canal; B, Distended central canal.

Fig. 5. Section of brain dog 1527. Natural size.

A, Marked congestion of brain stem. Histologically there were hemorrhages throughout, as well as hyperemia. Contrast this fairly normal cerebral cortex and lateral ventricles with Figs. 1 and 2.

Plate II

Fig. 6. Section of cord stained by the Marchi method. The black spots in the white matter are degenerated fibers.

Fig. 7. A portion of Fig. 6 under higher magnification. Showing the degenerated fibers by the Marchi method.

Plate III

Fig. 8. Section of cord showing perivascular infiltration x 450; A, Congested blood vessel; B, Lymphocytic infiltration in perivascular space.

Fig. 9. Cord of dog 1567, x 50, showing vacuolation of the fasciculus gracilis and cuneatus.

Plate IV

Figs. 10 and 11. Purkinje cells in advanced stages of chromatolysis dog 1742. x 450.

Fig. 12. Central canal filled with coagulum dog 1622. x 70.

Fig. 13. Motor cell showing chromatolysis dog 1742. x 450.

Fig. 14. Area of brain, region of Brachium pontis, dog 1622, x 70, showing vacuolation and cellular infiltration.

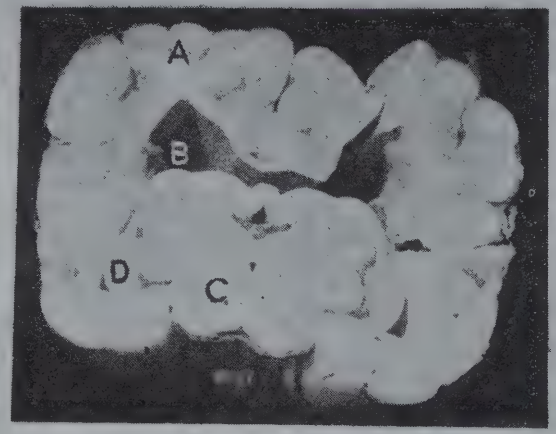


FIG. 1

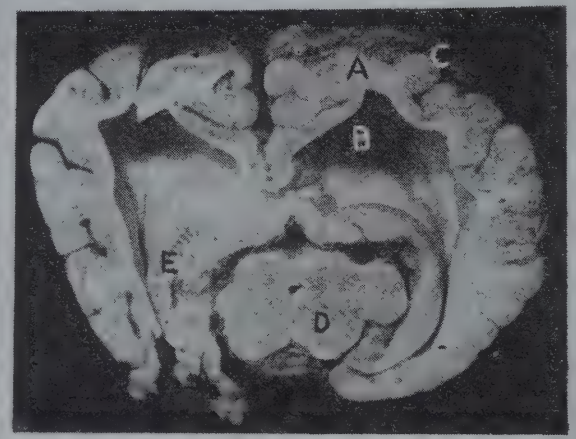


FIG. 2

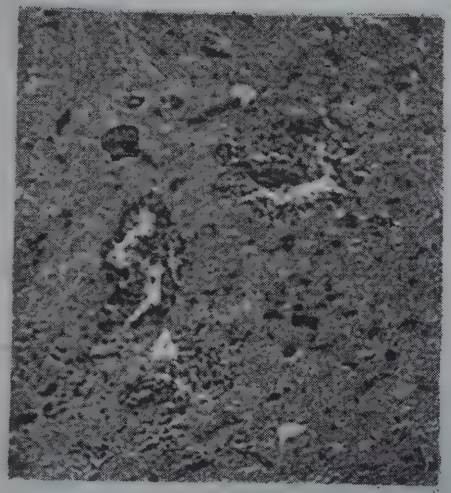


FIG. 3

FIG. 4

FIG. 5

Plate II.

FIG. 6

FIG. 7

Plate III.

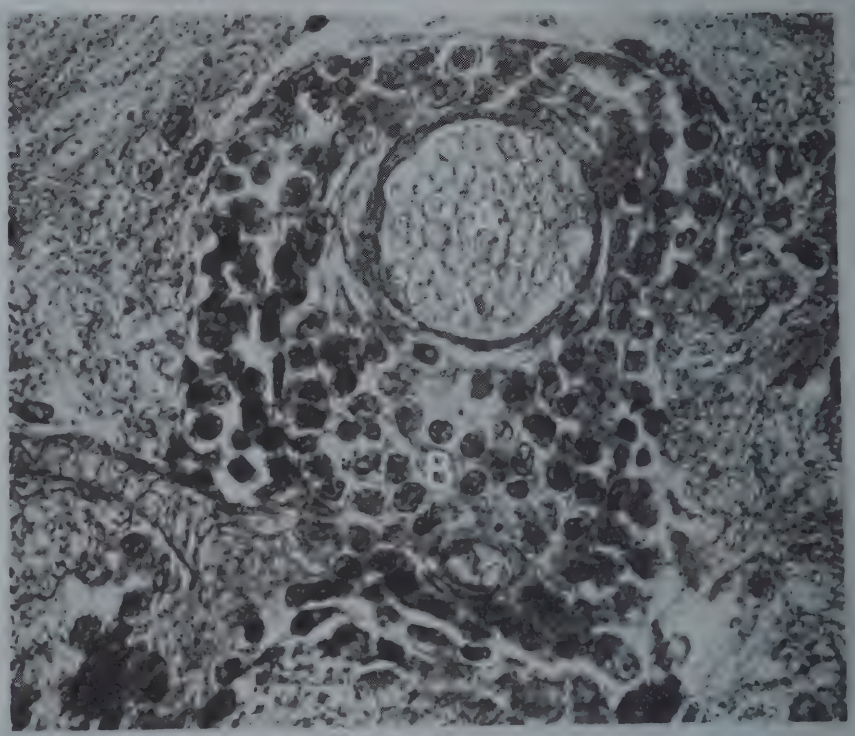


FIG. 8

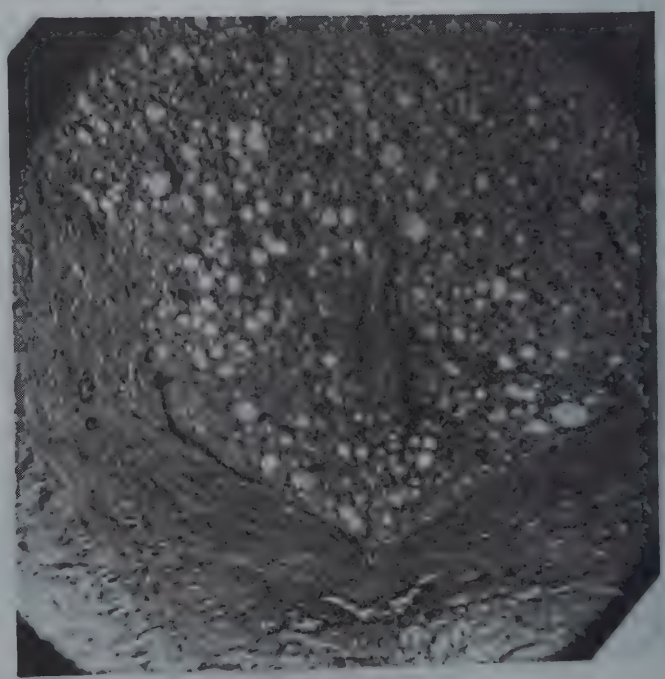


FIG. 9

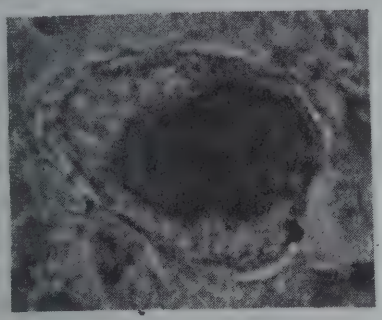


FIG. 10

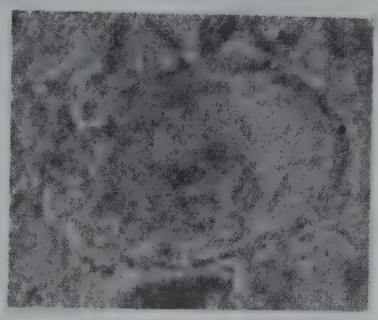


FIG. 11

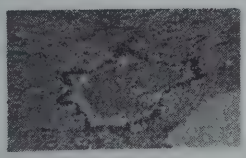


FIG. 12

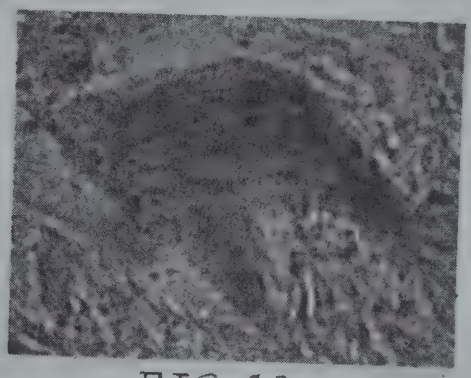


FIG. 13

FIG. 14

A STUDY OF SKIN TUBERCULOSIS*

C. M. CARPENTER AND S. A. GOLDBERG
Department of Pathology and Bacteriology

The attempt to eradicate bovine tuberculosis has developed many new problems for the men interested in the control of this disease. The Diagnostic Laboratory has from time to time in the past received specimens of bovine skin with lesions resembling tuberculosis. During the past year there has been a steady increase in the number of this type of lesion sent to the laboratory. On gross examination these lesions simulate tuberculosis, but in every case we failed to infect guinea pigs when extracts from these lesions were injected into experimental animals.

Inasmuch as the majority of these lesions came from cattle reacting to the tuberculin test they deserve more than passing notice. When a herd has been accredited there should be no more tubercle bacilli present to spread the infection. These obscure lesions may be responsible for some of the "no-lesion" cases. A small area of infection under the skin can be overlooked easily at an autopsy.

Traum[1] reported a disease in cattle under the title of "Lymphangitis in Cattle Caused by an Acid-Alcohol-Fast Organism." The lesions macroscopically and microscopically are similar in all respects to those we have observed.

Calmette[2] divides skin tuberculosis in man into four groups: lupus, tuberculous ulcers, tuberculous gummata, and tuberculous lymphangitis. We have observed all the above types in cattle, the most common being that which resembles lupus.

Calmette[2] states that bacilli are often lacking in the exudate from the ulcer surface and that they are usually attenuated in virulence. He also says:

"Although it is true that the inoculation into a guinea pig of a sufficiently large and properly ground-up fragment of lupus tissue gives rise always to the development of tuberculous lesions, the latter are in general benign; they progress slowly over a long period, manifest themselves only by a local adenitis near the point of inoculation, and the infection becomes generalized only after several months or even more than a year. This very evident attenuation of the bacillus cultivated in the dermic tissue explains why no one has ever been able to inoculate lupus from one human being to another."

In a discussion on bovine tuberculosis of the skin Calmette[2] says that this form of the disease is regarded as very rare in cattle. There are only some 20 cases reported. Inoculations indicate that the bacilli contained in these lesions have but a low degree of virulence in comparison with bacilli isolated from human lupus.

---

* The Cornell Veterinarian, Vol. XV (1925), p. 148.

Hastings and Beach[3] make the following statement in an abstract of an article entitled, "Positive Reactions to Tuberculin in Non-tuberculous Cattle."

"Fifty-four skin lesions from as many tuberculin-reacting cattle, showing no internal evidences of tuberculosis, have been examined microscopically, culturally and by animal inoculation. The cultures and animal inoculation tests were negative in every instance. Acid-fast bacilli were found microscopically in 15, or 27 per cent, of the lesions."

One of us (S. A. G.) studied the histological material of Dr. Traum and also a large number of cases of lupus vulgaris in man. The latter work was done at the College of Physicians and Surgeons, Columbia University. The lesions in Traum's material, in the human material, and in our own were found to be practically identical.

We have studied up to the present 30 cases of this type of lesion. The material has been collected from all parts of New York State, but we do find that this infection is more prevalent in certain localities than in others. That this condition is not rare is evidenced by the following extract from a letter received at the laboratory:

"These cows reacted to both the intradermic and opthalmic tests. In my work here I have had at least 40 such cases, and I can remember only two or three with the characteristic bunches which have passed these tests without a sign of a reaction, all the rest having well defined reactions to both tests. Very often these reactions seem to come on the retest, the herd having passed one or more clean tests."

As a rule the lesions are found on the extremities of cattle, although they may appear anywhere on or underneath the skin. We have observed lesions on the genitalia of bulls, especially in the connective tissue of the sheath and prepuce, that are identical, macroscopically and microscopically, with the lesions found in the skin and subcutis of the extremities. Animals of all ages are affected, but heifers or young cows seem to be most susceptible.

The history of several of our cases shows that the nodules developed on the extremities following a wound such as a laceration from an entanglement with barbed wire. In one case the cow had been bitten on the leg by a dog, following which the first nodules developed and then continued to spread up the limb. These histories are very suggestive of a wound infection. Undoubtedly there are many small abrasions on the legs of cattle that pass unnoticed but which would serve as an ideal place for the entrance of the infection.

The nodules vary in size from a pin-point lesion to several centimeters in diameter. Many of the larger nodules have spread over a comparatively large surface of the skin while in other cases smaller nodules appear in a chain or cord. On section the nodules may be caseous or calcareous and may have undergone liquefaction necrosis. If liquefaction has occurred the pus generally has the

appearance and consistency of a light colored peanut butter in which there are sometimes deposits of lime salts.

Smears from the lesions may or may not show acid-fast bacteria. In the cases which we have studied failure to observe acid-fast organisms has been the rule. In some cases bodies were seen which retained the carbol-fuchsin after being treated with acid alcohol, but in very few cases were we positive that these bodies were acid-fast bacilli. Histological sections made from the lesions usually failed to show acid-fast bacilli when they were stained by the Ziehl-Neilson and Schultz-Tigge methods.

Microscopically the young lesions are very interesting because the development usually begins with an infiltration of lymphocytes and endothelial cells around a capillary or blood vessel. As the lesion becomes more advanced there is a degeneration of the blood vessel and other cells in its proximity and finally there is developed a tubercle with a necrotic center showing giant cells, lymphocytes and endothelial cells in the peripheral zone (See plates). Intermingled with these cells are many blood vessels which make the peripheral zone comparatively vascular. These lesions differ from a typical tuberculous nodule in such organs as the spleen, lymph glands and lungs in that the young lesion starts its development around a blood vessel and because of the production of many blood vessels in the peripheral zone of the nodule. The explanation for this variation may be due to the fact that the skin produces a tissue reaction different from that of the parenchymatous organs.

Experimentally we have failed in every attempt to infect guinea pigs, rabbits and chickens with extract from these lesions, even though in some cases many acid-fast bacilli were present. We have used birds because some have suggested that it is the avian type of tubercle bacillus that produces the lesions in question. Calmette[2] has explained that inasmuch as the skin and subcutis are unfavorable locations for the tubercle bacillus to develop, the virulence is markedly decreased.

It has been frequently observed that a virus becomes attenuated or loses its virulence when placed in dermic tissue. When the virus of contagious pleuropneumonia is injected intradermally it fails to produce the disease. More recently an immunity to anthrax has been developed by Besredka[4] in guinea pigs and rabbits by injecting the anthrax bacillus intracutaneously. Brocq-Rousseu and Urbain[5] have produced an immunity in horses by injecting this organism in the same manner.

EXPERIMENTAL

Some of the above skin lesions from case 9815, showing large numbers of acid-fast bacilli, were placed beneath the skin just above the knee of a cow. A tuberculin test made on the day the animal was inoculated was positive. For several days following the transplantation there was a marked edema of the forearm due to the wound produced. This healed readily and the leg returned to normal within two weeks. The cow was slaughtered

three and one-half months later. The region in which the tissue had been transplanted was apparently normal.

One-tenth of a mil of a suspension of bovine tubercle bacilli was injected intradermally on the external surface of each of the front legs below the knee of a two-year-old heifer (243) which was negative to the subcutaneous, intradermal and ophthalmic tests. The suspension used gave a reading of three, on the Gates[6] apparatus. An incision three centimeters long was made in the skin just above the metacarpo-phalangeal joint on each front leg. Some of the same suspension used above was swabbed in the opening made by the incision and left to heal. Little nodules developed at the point of injection while about one-half centimeter on each side of the incisions quite an extensive amount of more or less nodular cicatricial tissue formed. Seven weeks later a lesion of each type was incised. Half was sectioned while an extract was made of the other half of each lesion and injected into guinea pigs. The lesions from both sides showed tubercles practically identical with those observed in natural infections. The guinea pig injected with the extract from the lesion on the right front leg died with generalized tuberculosis eight weeks after inoculation. The guinea pig injected with an extract from the nodule on the left side is living but recently gave a positive reaction to the intradermic tuberculin test. He shows no evidence of enlarged inguinal lymph glands and has not decreased in weight.

The other two nodules which developed in the heifer have been left in the animal and seem to remain about the same size as they were one month after the inoculation. The nodules will be removed and examined later. The animal now gives a good reaction to the intradermic and ophthalmic tuberculin tests.

Although the nodules produced with the culture in the heifer developed tuberculosis in guinea pigs, it must be remembered that the culture was in the skin only a comparatively short period of time (7 weeks). In the natural infection the lesions probably remain a long time before they are observed, and after they have been detected it is often a question of years before an autopsy is made and the lesion examined. Unquestionably a greater number of the organisms were injected than naturally enter the skin if this condition is the result of wound infection. On the other hand the similarity of the lesions produced artificially and naturally may be due to the fact that they are both caused by acid-alcohol-fast organisms which are closely related.

SUMMARY

1. The cutaneous lesions described in cattle are identical with skin tuberculosis (*Lupus vulgaris*) in man.

2. The inoculation of a small amount of a culture of the bovine tubercle bacillus into the skin of cattle produces lesions identical with those that have come to our attention from natural infection.

3. Taking these thirty cases as a basis, the percentage of reactors to the skin form is approximately the same as the percentage of reactors to tuberculosis in other organs.

We are indebted to Drs. D. H. Udall and R. R. Birch for the cattle used in this work. We are also grateful to Dr. C. C. Walker of Bath, N. Y., and to all other veterinarians who have furnished us with material and data for this investigation.

BIBLIOGRAPHY

1. TRAUM, J. J. A. V. M. A. 1916, xlix, 254.
Ibid., J. A. V. M. A. 1919, lv, 639.
Ibid., Cornell Vet. 1923, xiii, 240.
2. CALMETTE, A. Tubercle Bacillus Infection and Tuberculosis in Man and Animals, 1923.
3. HASTINGS AND BEACH. Abstract distributed at meeting of S. A. B. 1924, Washington, D. C.
4. BESREDKA, A. Comp. rend. Soc. de biol. 1920, lxxxiii, 769.
Ibid., and de Trevise. Ann. de l'Inst. Pasteur, 1922, xxxvi, 562.
5. BROCQ-ROSSEU AND URBAIN. Ann. de l'Inst. Pasteur, 1924, xxxviii, 268.
6. GATES, F. L. J. of Exp. Med., 1920, xxxi, 105.

TABLE 1

Results of tuberculin tests on animals affected with skin tuberculosis and laboratory examinations on cutaneous lesions removed from them at autopsy. No other tuberculous lesions were found on post mortem examination.

Laboratory accession number	F I	S I	S II	B I	B II	3898	3941	7634A	7634B	7853	8232	9434	9483	9497	9510	9706	9815	9861	9902	9929	10016-1	10016-2	10016-3	10060	10092	10181	10238	10245	10252	10264
Subcutaneous tuberculin	+	+	+	+	+	...	+	6*	6	+	...	...	...	...	...	...	+	...	4	...	...	...	...	—	...	...	...	...	...	...
Intradermal tuberculin	...	...	...	...	...	...	...	+	+	...	+	+	+	+	+	...	...	+	4	3	+	+	+	+	+	+	+	+	+	+
Ophthalmic tuberculin	...	...	...	...	...	...	...	...	...	...	+	+	+	+	+	...	...	+	4	3	+	+	+	...	...	+	+	+	? +	+	+ +
Acid-fast bacilli	...	...	+	+	+	+	—	—	—	—	—	—	?	+	—	?	+	?	+	—	+	+	+	...	...	—	—	—	—	—	—
Guinea pig inoculation	—	—	—	...	...	...	—	...	...	—	—	—	—	—	—	—	—	...	—	—	—	—	—	...	—	—	—	—	—	—
Rabbit inoculation	...	...	...	...	...	...	...	...	...	...	...	—	...	...	—	—	—	...	—	—	—	—	—	...	—	—	—	—	—	—
Fowl inoculation	...	...	...	...	...	...	...	...	...	...	...	—	...	—	—	—	—	...	—	—	—	—	—	...	—	—	—	—	—	—

* Figures represent the number of times the test was applied.

? Indicates that acid-fast bodies were observed in smears made from the lesions but they were not typical of the tubercle bacillus.

DESCRIPTION OF PLATES

Plate I

Fig. 1. Subcutaneous tissue cow 3898. x ½.
A. Area of necrosis.
B. The lesion running in lines indicating spreading along lymphatics.
Fig. 2. Calcified nodules skin cow 3941. x ¼.
A. Area of calcification.
Fig. 3. Skin foot cow F I. x ½. Showing subcutaneous caseous and calcified nodules.

Plate II

Fig. 4. Penis bull showing submucous caseous nodules (after Williams).
Fig. 5. Subcutaneous nodules skin cow. x ½.
Fig. 6. Vascular proliferation and perivascular infiltration. Experimental cow 243. x 70.

Plate III

Fig. 7. Vascular proliferation and perivascular infiltration. Cow 9815. x 300.
Fig. 8. Lesion cow 9815. x 50.
A. Perivascular infiltration.
B. Nodule showing a giant cell.
Fig. 9. Calcified lesion cow 9434. x 50.
A. Perivascular infiltration.
B. Calcified area.

Plate IV

Fig. 10. Perivascular infiltration. Experimental cow 243. x 225.
A. Degenerated vessel suggesting formation of giant cell.
B. Lymphocytic infiltration.
Fig. 11. Lesion right leg. Experimental cow 243. x 225.
A. Giant cells.
B. Perivascular infiltration.
Fig. 12. Nodule skin homo 1005. x 100.
A. Vascular prolifieration and perivascular infiltration.
B. Giant cells.

Plate I.

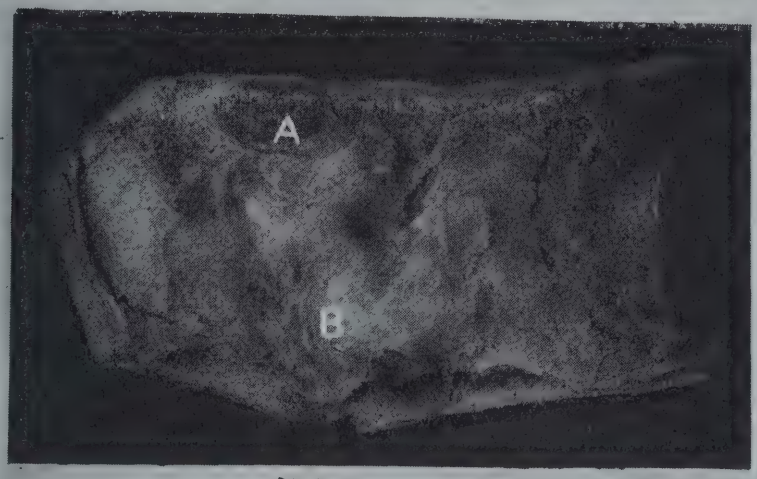


FIG. 1

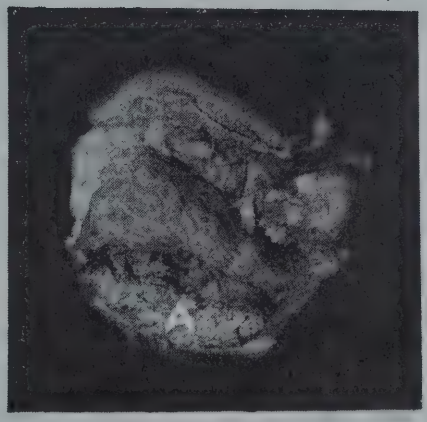


FIG. 2

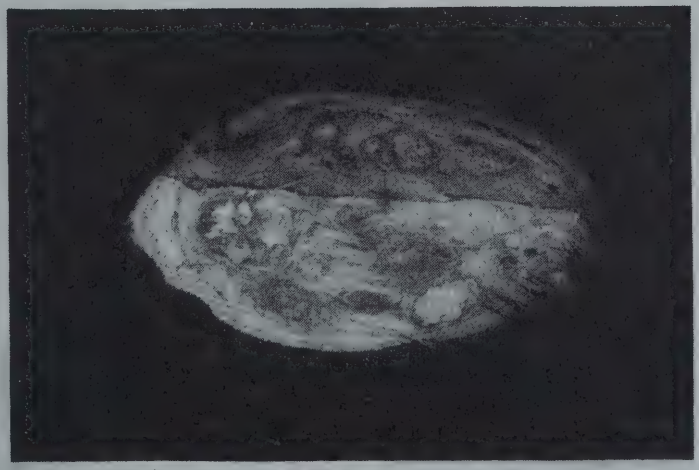


FIG. 3

Plate II.

FIG. 4

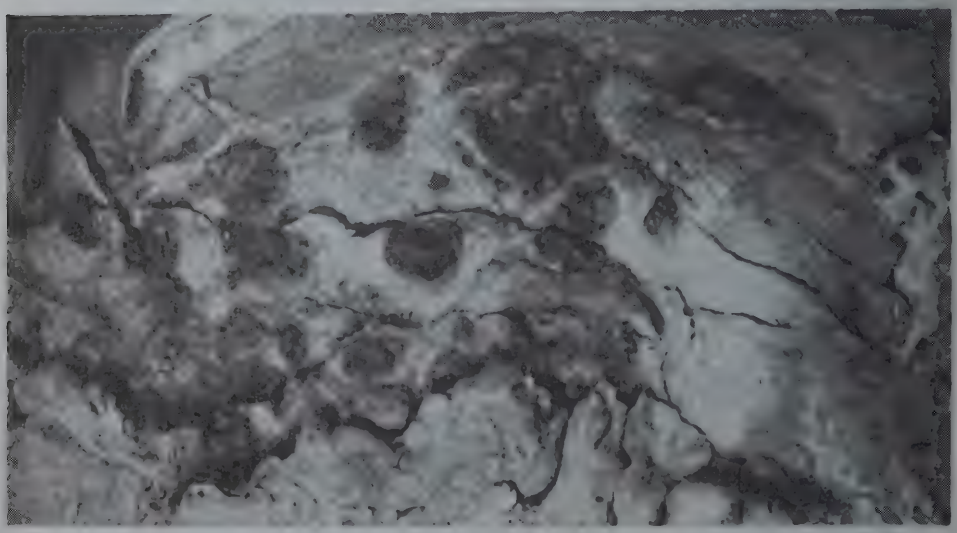


FIG. 5

FIG 6

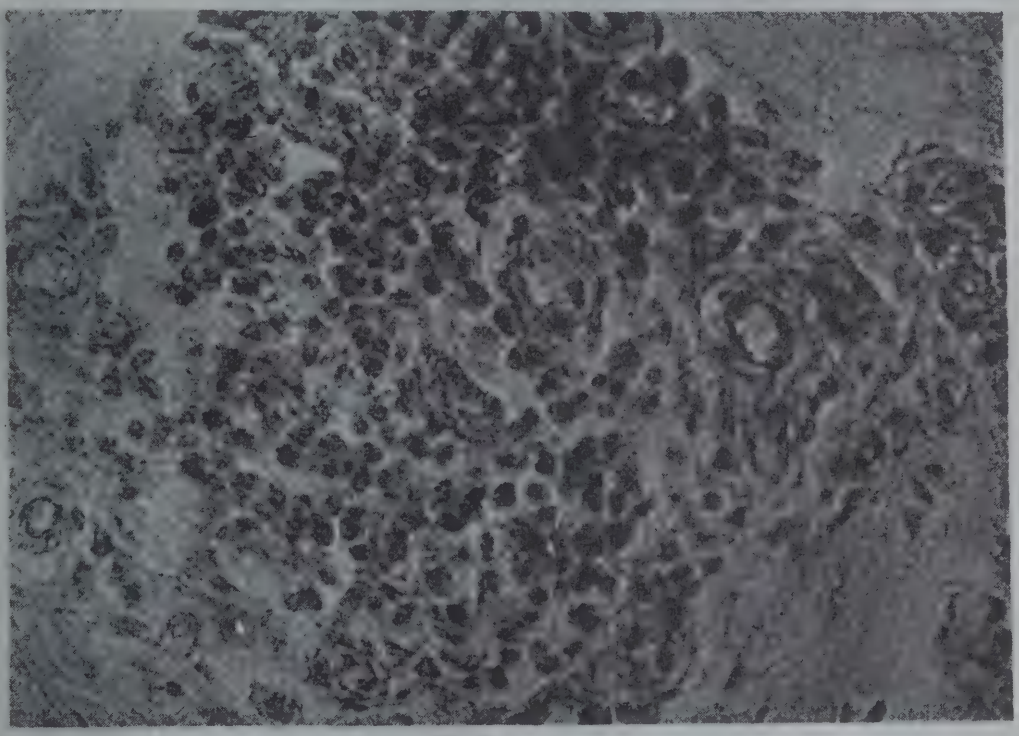


FIG. 7

FIG. 8

FIG. 9

FIG. 10

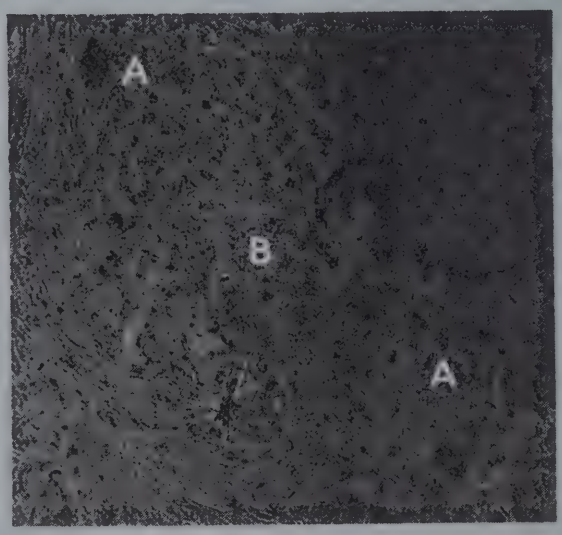


FIG. 11

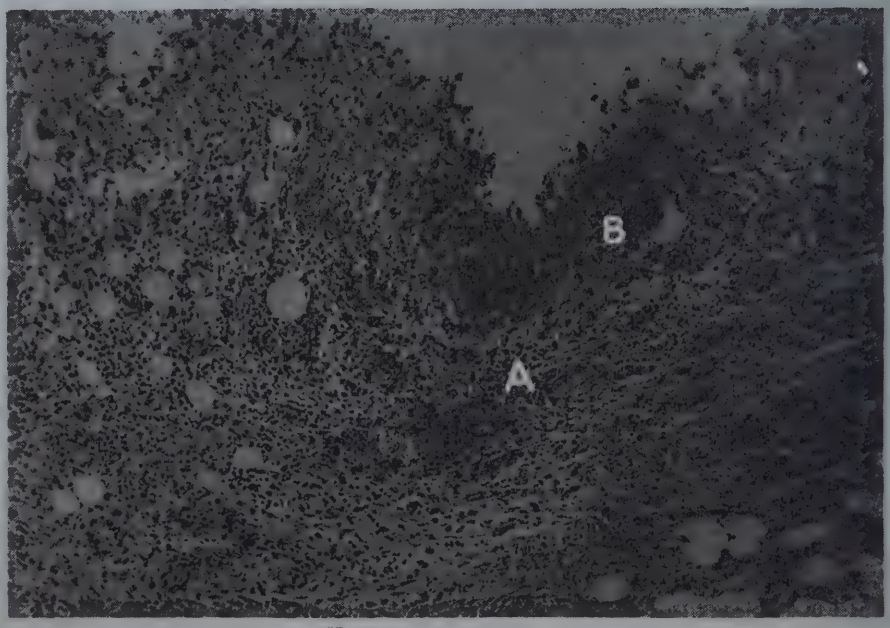


FIG. 12

FURTHER OBSERVATIONS ON THE BLOOD AND URINE OF THE HORSE IN AZOTURIA

C. E. HAYDEN

Department of Physiology

Chemical methods in the study of the blood[1] have recently taken a rather broad scope. They have included methods for blood volume, the blood proteins, serum albumin and globulin, improved methods for the hemoglobin estimation; methods for the determination of nonprotein nitrogen and its individual components, urea, creatinine, uric acid, amino acids, creatinine and ammonia; methods for sugar and the lipoid constituents,— fat, lecithin and cholestrol; methods for the mineral constituents, chlorides, phosphates, calcium, magnesium, potassium, sodium and iron; methods for the blood gases, carbon dioxide, carbon monoxide and oxygen; and methods for the hydrogen ion, the acetone bodies, phenol and such enzymes as diastase, catalase, etc.

These methods have yielded helpful information in regard to various human diseases and given new points of view regarding many disorders.

To further aid in the physiology and pathology of the horse Hayden and Tubangui[2] studied the composition of the blood from the standpoint of urea, uric acid, total nonprotein nitrogen, creatinine and sugar. Holt and Reynolds[3] have reported upon the determination of these substances in the blood of about 150 horses.

In addition to the substances named Holt and Reynolds[3] determined the hemoglobin content of the blood.

The average obtained for the blood of the normal horse is as follows:

Mgms. per 100 mls., Blood

	Sugar	Total nonprotein nitrogen	Creatinine	Urea	Uric acid
Hayden and Tubangui	102.9	33.9	1.81	18.71	2.45
Holt and Reynolds	109.	25.82	1.4	14.81	2.76

In the consideration of these averages it will be noticed that the comparison is rather close with the exception of nonprotein nitrogen. The average of 33.9 mgms. was taken from readings of a much smaller number of animals. It is possible that readings from a large number of animals tend to lower the average. Uric acid figures for the blood of the herbivorous animals needs review. Methods for the determination of uric acid have changed and it is possible that revised readings will lower the average in both groups. It seems tenable however that even though present figures be too high the comparative value still exists.

There is a difference of 4 mgms. in the urea averages in the two

tables. In the author's opinion the urea would need to rise to 20 mgms. or above before it indicated any degree of retention. Further readings on the normal blood of the horse are desirable in order to check present data and to keep up with the refinements in technic especially in the determination of uric acid and sugar.

Holt and Reynolds[3] include a table of determinations in diseased conditions but none of the readings are for the blood of the horse in azoturia.

BLOOD FINDINGS

Mgms. per 100 mls., Blood

CASE NUMBER	Sugar	Total non-protein nitrogen	Preformed creatinine	Urea	Uric acid	Chlorides
1	78.1	35.7	1.21	8.82	3.6	489.
2	217.	31.5	1.2	7.55	2.97	478.5
3	170.	33.	1.2	9.15	3.2	528.
4	85.9	42.9	2.73	12.8	5.18	396.
5	45.5	41.8	1.5	10.8	2.7	429.
6	97.	38.10	0.96	15.6	0.389	495.
7	112.5	18.33	1.17	22.9	4.04	412.5
8	110.7	38.10	1.18	19.6	2.14	313.5
9	94.6	40.8	1.18	13.	Trace	445.5
Average	112.3	35.58	1.37	13.36	2.679	443.
Average twelve, four samples	121.9	38.24	1.74	17.62	2.808	.....
Average both reports	117.1	36.91	1.55	15.49	2.743	443.

In this report of 9 cases there are 2 high sugar readings. The average is within normal. No great disturbance in carbohydrate metabolism is indicated by the average reading. The two individual cases do show a high sugar reading but it is possible that this may be accounted for by the nervous condition of these two cases.

Nonprotein nitrogen and its component parts; urea, uric acid, and creatinine are very close to normal when compared with the normal reading of Hayden and Tubangui.[2] No marked nitrogen retention is indicated by average or even individual readings.

Chlorides, as a representative of the salts of the blood, have been determined. The average for the nine samples of this report is 443 mgms. per 100 mls., blood. The normal reading for the blood of the horse, using the same technic, is not yet available. When compared with the normal reading of 516.7 mgms. for the blood of the cow obtained by Hayden and Sholl[4] it does not seem high. The average reading for sugar, nonprotein nitrogen, creatinine, urea, and uric acid for 21 cases of azoturia does not depart greatly from the normal. These figures fail to support the theories that there is marked renal impairment or that the blood is loaded with the so-called leucomaines or extractives of the blood.

URINE FINDINGS

The addition of urine data has not changed the average given by Hayden[5] in the last communication. The specific gravity falls within normal limits. The reaction is consistently alkaline. The

color is from normal to the coffee colored urine which is characteristic of the urine in azoturia. Urines which are near normal in color give both chemical and spectroscopic tests for blood. Hemoglobin seems to be a general constituent in azoturia. The elimination of salts such as chlorides, sulphates and phosphates is normal. The elimination of urea and uric acid is normal. The ready elimination of these substances does not indicate any marked degree of renal impairment. Indican is not eliminated in undue quantities. Albumin and sugar are found in some samples. Albumin has occurred in quantities up to 6.09 grams in 1000 cc. of urine, sugar as much as 4.1 grams in like amount of urine. A trace of bile is met with in some samples. Occasional hyaline and epithelial casts are found in occasional samples. Hemoglobinuria uncomplicated by hematuria is characteristic of the disturbance. No crystals of any importance are found on microscopic examination.

Conclusions

1. There is a moderate average increase in nonprotein nitrogen and sugar in the blood in azoturia.
2. Individual cases tend to raise the averages. These cases are not always the worst clinical cases.
3. The increase in extractives is too slight to justify the claim that the blood is loaded with these substances. There is no indication of an uremia.
4. The hyperglycemia and glycosuria met with in such slight degree do not justify the claim that azoturia is due to disturbed carbohydrate metabolism.
5. Chlorides have averaged 443 mgms. for 100 mls., blood in 9 cases of azoturia. Normal figures by the same method are not available for the horse.
6. Blood and urine changes noted are secondary rather than primary to this marked clinical disturbance.

Acknowledgments

Acknowledgments are due Dr. J. N. Frost, Dr. E. R. Cushing and Dr. M. G. Fincher for samples of blood and urine furnished. Dr. L. B. Sholl participated in part of the blood readings and his assistance is gratefully acknowledged.

References

1. Myers, V. C., Practical Chemical Analysis of the Blood. Second Edition, 1924.
2. Hayden and Tubangui, Studies on the Normal Blood of the Domesticated Animals, Report of the New York State Veterinary College at Cornell University, 1920, 181–192.
3. Holt and Reynolds, Blood Chemistry of the Horse, Jour. Am. Vet. Med. Assoc., lxv., 732–736.
4. Hayden and Sholl, A Study of the Extractives of the Blood of the Cow, Report of the New York State Veterinary College at Cornell University, 1923–1924, 102–110.
5. Hayden, C. E., The Blood, Urine and Tissue Juices in Azoturia, Cornell Veterinarian, xiv, 158–165.

THE BLOOD AND URINE OF THE COW IN MILK FEVER

A SECOND REPORT

C. E. HAYDEN

Department of Physiology

Data upon the composition of the blood and urine of the cow in milk fever have been submitted by Hayden and Sholl[1] for 14 cases of the disorder. Only three samples of urine were analyzed as against 14 samples of blood from so many cases of milk fever. The literature covering milk fever, and the theories therein set forth, have been reviewed in the report by Hayden and Sholl.[1]

Since the time of the first report there have been more recent publications touching on the toxemias of pregnancy in both the bovine and human families.

W. L. Williams, D. H. Udall, J. N. Frost et al.[2] say that they were not prepared to find that in milk fever there were, so far as shown by their studies, ample utero-fetal lesions to offer a plausible explanation for the clinical phenomena. They refer to the views of Schmidt that the malady was due to a toxemia and indicate that the utero-fetal lesions could be responsible for such a toxemia.

These same authors say that the study of milk fever, while important in the better prevention and control of the disease in cows, has far greater possibilities in relation to medical science as a whole. One of the great and difficult problems facing the human obstetrist is parturient eclamptic diseases, or the toxemias of pregnancy. There has long been suspected a relationship between the toxemias of pregnant women and milk fever or parturient paresis of cows, but there have always been in both fields serious conflicts of opinion and a great scarcity of illuminating data.

Myers[3] reviews data presented by Killian and Sherwin,[4] Caldwell and Lyle,[5] and Williams[6] and says that recent data on the chemistry of the blood in the toxemias of pregnancy, eclampsia and hyperemesis, have considerably modified the older views regarding these toxemias, chiefly because they have given us definite information regarding renal function. In normal human pregnancy the values for the nonprotein and urea nitrogen are slightly low and there is some decrease in the CO_2-combining power during the last months, but otherwise the blood is essentially normal. With true eclampsia, also hyperemesis, there appears to be only slight impairment in renal function, contrary to older assumptions. The nonprotein nitrogen is moderately increased, but the urea nitrogen is at the low normal limit or decreased, constituting 15 to 40 per cent of the nonprotein nitrogen instead of the normal 50 per cent. As a rule the uric acid is definitely increased, due apparently to a slight impairment in renal function. Taking the CO_2-combining power as the index there is a moderate or severe acidosis in all cases.

Some cases show high blood chlorides and marked edema, others a slight but definite hyperglycemia. Judged both from the chemical changes in the blood and the clinical symptoms prompt improvement follows the evacuation of the uterus in most cases. Toxemias do occur in which nitrogen retention exists but it seems probable that in most cases this is due to a true nephritis, which may have antedated the pregnancy. A high creatinine in such cases furnishes an unfavorable prognosis.

Menig[7] emphasizes decalcification and the production of acidosis in discussing the causes of milk fever. He says that the urine is scanty and of high concentration preceding the attack. Renal insufficiency is dwelt upon. He indicates alkaline therapy in the toxemias of bovine pregnancy.

Blood Findings

Blood samples have been obtained from 14 different cases of milk fever. Samples after udder inflation and general treatment have been obtained from only two cases. This is to be lamented, for more data after treatment are very desirable. The animals have been pure breds and grades, they have been indicated as being from 4 years in age to aged in some instances. The cases have been straight milk fever in most instances and in others have been complicated with metritis or retained placenta or both. They, in common with the fourteen reported upon in the preceding report, represented a good range of the toxemias of pregnancy. The cases reported here have come from about the same herds as those from which the samples of the preceding report came.

FIRST SAMPLE OF BLOOD OBTAINED

Mgms. per 100 mls., Blood

CASE NUMBER	Sugar	Total non-protein nitrogen	Preformed crea-tinine	Urea	Uric acid	Chlorides
15	70.7	43.80	0.985	13.5	3.32	495.
16	74.6	42.	1.13	12.9	0.93	462.
17	84.	46.80	.....	20.	1.5	495.
18	111.1	37.5	1.11	18.4	1.84	478.5
19	140.5	36.	1.12	34.6	0.186	429.
20	89.1	37.6	1.24	18.8	1.8	445.5
21	88.1	27.	1.42	17.04	1.87	495.
23	110.7	55.53	1.12	29.7	2.01	.....
24	92.9	36.90	1.28	15.1	3.01	.....
25	123.2	30.9	1.27	12.2	2.33	462.
26	130.	39.	1.29	15.6	3.7	478.5
27	63.	41.4	2.14	18.3	2.5	544.5
28	50.	27.30	1.24	14.8	2.22	429.
Average	94.45	38.59	1.279	18.53	2.093	474.

SECOND SAMPLE OF BLOOD OBTAINED

Mgms. per 100 mls., Blood

CASE NUMBER	Sugar	Total non-protein nitrogen	Preformed crea-tinine	Urea	Uric acid	Chlorides
15	97.08	37.8	0.99	13.7	2.01	511.5
17	64.	36.	.....	14.8	1.59	528.
Average	80.54	36.9	0.99	14.25	1.80	519.75

TABLE OF AVERAGES

Mgms. per 100 mls., Blood

	Sugar	Total nonprotein nitrogen	Preformed creatinine	Urea	Uric acid	Chlorides
Normal	51.75	30.11	1.37	11.18	2.06	516.1
Twenty-seven first samples in milk fever	95.82	36.88	1.31	17.08	2.25	472.7
Nine second samples in milk fever	72.76	37.73	1.11	14.12	2.19	515.62

The normal average is taken from the report by Hayden and Sholl.[8] Fourteen of the first samples and seven of the second samples which enter into the averages are included in the report by Hayden and Sholl.[1] An average of the first sample of blood taken in milk fever indicates a rather marked hyperglycemia. The second set of samples which are taken after treatment still shows hyperglycemia but there is quite a marked decrease in the sugar content of the blood after udder inflation.

There is an increase in total nonprotein nitrogen in both sets of samples. The average for the second set is higher than that of the first. There is an increase in the urea in both sets but in this case the first set shows the greater increase. Urea is a part of the total nonprotein nitrogen and it makes up a larger portion of the nonprotein nitrogen in milk fever than it does in the normal animal. Killian and Sherwin[4] found it decreased slightly in the blood of human eclampsias.

There is a slight average increase in the uric acid readings. The average increase of these nitrogenous end products does not seem to indicate any considerable impairment of renal function. Reference to individual cases in the tables would indicate a greater impairment in some animals than in others. Creatinine is the extractive most easily eliminated by the kidneys. Its retention in any quantity would point to serious renal disturbance. The average of our readings shows a decrease rather than a retention. Creatinine eliminated in the urine has not been determined and so the blood and urine figures cannot be compared.

There is a decrease in chlorides in the first series of samples obtained. The second series gives average figures which are quite close to that of the normal.

URINE FINDINGS

Average for 12 samples

	Normal	Low	High	Average
Specific gravity	1015–1045	1014	1042	1030
Reaction	Alkaline	Alkaline	Acid	6 samples; acid
Color	Yellowish	Yellowish	74°	6 samples, alkaline
Translucency	Slightly turbid	Clear	Red	Normal
Solids	52–144	32.62	Turbid	Slightly turbid
Chlorides	3–5	Trace	97.8	69.90
Sulphates	2–3	0.6	18.2	3.79
Phosphates	Trace	None	14.7	3.41
Urea	15–35	5	3.9	0.54
Uric acid	.20–.40	None	35.	17.25
Indican	Little	None	0.68	0.23
Albumin	None	Trace	Medium	Little
Sugar	None	1.1	0.84	0.213
Bile	None	Slight trace	25.	8.13
Hemoglobin	None	None	Trace	8 samples normal
Epithelial cells	Occasional	Occasional	None	None
Leucocytes	None	None	Large number	Few
Blood	None	None	Few	Occasional
Casts	None	None	Numerous red cells	7 samples, no red cells
			Numerous epithelial	10 samples, none
CRYSTALS				
Uric acid	None	None	Few	Occasional
Oxalates	None	None	None	None
Triple phosphates	None	None	None	None
Urates	None	None	None	None

Fermentation was positive in 4 samples. The phenylhydrazine test yielded lactozone crystals in five samples and dextrozone in two. In one sample in which fermentation was marked neither type of crystal was observed. In another giving marked fermentation lactozone crystals only were identified. In some of the samples it appears that lactose was present. In others dextrose seems to have been the main reducing substance. It is possible that lactose and dextrose were both present in at least two of the samples. In some samples neither fermentation was brought about nor were crystals formed. There are at times reducing substances present in the urine which are not sugars and this may have been true of some of the samples tested in this series.

Acetone bodies are taken in human urological work as an indication of acidosis. The tests for acetone and diacetic acid have been applied to these samples. They were both indicated in one examination and that was in the same sample of urine. Even though the sugar has averaged 8.13 grams to the 1000 of urine these bodies do not seem to be present in consistent pathological quantities. This does not preclude the very evident possibility of

an acidosis, for the normal alkaline reaction has been changed to acid in at least 6 of the samples.

A constant albuminuria and glycosuria seem to be indicated by the data of the twelve samples examined. The albumin may be taken as an indication of a slight renal impairment. Neither the blood nor the urine findings would indicate a marked impairment. Casts were present in two of the samples examined. It seems that they might be found more often if the renal impairment were sufficient to cause milk fever. The glycosuria may be expected when the apparent hyperglycemia which characterizes cases before treatment is considered.

The average specific gravity and total solids do not indicate urines of high concentration. The elimination may be slight but there does not seem to be concentration as is expressed by Menig.[7] There may be an acidosis. This is shown to be the case in eclampsia but it remains to be demonstrated in bovine parturient paresis.

CONCLUSIONS

1. There are hyperglycemia and glycosuria in milk fever.

2. The hyperglycemia begins to decrease with udder inflation and accompanying treatment.

3. The hyperglycemia may be a reflex of the extreme nervous reaction evident before treatment.

4. Nonprotein nitrogen and urea are both above normal. Average increase indicates only slight renal impairment.

5. Uric acid in the blood is not increased in the same proportion as it is in eclampsia. There is a very slight increase but not enough to indicate renal disturbance.

6. Chlorides in the blood decrease somewhat before treatment but return quickly to normal in average cases.

7. Glycosuria is a constant feature. It is due in some cases to lactose, in others to dextrose and in others to a combination of these two reducing sugars.

8. The urine is of average specific gravity and average concentration and shows a constant albuminuria. There is evidence of slight impairment of renal function.

9. Blood and urine findings in the eclampsia of human pregnancy and parturient paresis show rather close comparison. The points of similarity warrant further comparative study.

ACKNOWLEDGMENTS

Acknowledgments are due Dr. M. G. Fincher and Dr. E. R. Cushing who have furnished the samples of blood and urine which are the basis of this report.

REFERENCES

1. HAYDEN, C. E. AND L. B. SHOLL. The Blood and Urine of the Cow in Milk Fever. Report of the New York State Veterinary College. 1923–24, 91–101.
2. WILLIAMS, W. L., D. H. UDALL, J. N. FROST, S. A. GOLDBERG, E. R. CUSHING, M. G. FINCHER AND M. H. MABEY. Studies in Genital Disease. Cornell Veterinarian. October, 1924.
3. MYERS, V. C. Practical Chemical Analysis of the Blood. Second Edition, 1924.
4. KILLIAN, J. A. AND SHERWIN, C. P. Some Chemical Studies in Normal and Abnormal Pregnancies. 1. Significant Chemical Changes in the Blood in the Toxemias of Pregnancy. American Jour. Obst. and Gynec. 1921, 11, 6–16.
5. CALDWELL, W. E. AND LYLE, W. G. The Blood Chemistry in Normal and Abnormal Pregnancy. Amer. Jour. Obst. and Gynec. 1921, 11, 17–34.
6. WILLIAMS, J. W. Increased Amount of Uric Acid in the Blood in the Toxemias of Pregnancy. Jour. Am. Med. Assoc. 1921, lxxvi, 1297–1299.
7. MENIG, O. Auto-Intoxication of Advanced Pregnancy. Jour. Am. Vet. Med. Assoc. lxxvi, 711–721.
8. HAYDEN, C. E. AND SHOLL, L. B. A Study of the Extractives of the Blood of the Cow. Report of the New York State Veterinary College. 1923–1924, 102–110.

CASE REPORT

ILLUSTRATING THE VALUE OF THE X-RAY IN DIAGNOSIS

E. M. CURLEY
Captain Veterinary Corps

During the forenoon of December 1, 1924, horse Number 3, a black gelding, aged 10 years, while tied to the picket line of the R. O. T. C. stables at Cornell University, Ithaca, N. Y., was kicked by another horse. The plight of the animal was immediately discovered by the stable sergeant and the attending veterinarian notified.

A diagnosis of a wound lacerated involving the posterior surface of the right hock, and a wound of the same nature on the external surface of the same structure, but more severe than the former, was made. The former wound from all appearances involved the joint cavity, that is, an open joint of the articulation (fixed) between the cuboid and the outer splint bone. While the animal evidenced pain on palpation and during locomotion, still it was not severe enough to warrant a diagnosis of an open joint or even a fracture. However, both conditions were suspected. During the succeeding forty-eight hours the animal assumed the recumbent position practically all the time, the appetite remained good, temperature normal, but the entire tarsal joint became very much distended. Hot fomentations were administered several times a day and the wound antiseptically treated. Very little improvement, if any, was noted.

At this point it would be well to say a few words in reference to the anatomy of this part. The ligaments involved on the outside of the hock are the long lateral ligament (Sisson), which runs from the external tuberosity of the femur to the articular head of the external small metatarsal bone (splint), and the plantar ligament from the head of the calcaneum to the articular head of the external small metatarsal bone. The capsular ligament, which was much inflamed, ends about one-half inch below the articular surface of the splint bone. No doubt the joint was opened by the original blow. It must be obvious that the capsular ligament was opened at the same time, producing, as commonly referred to, an open joint, which according to our veterinary diagnostic nomenclature should be recorded as "wound involving joint cavity."

On the 4th of December the animal was taken by ambulance to the Veterinary Hospital in connection with the Veterinary College. At this point I wish to call the reader's attention to Plate 1, which has no connection with the case, but merely to point out an X-ray picture of the outline of a sound tarsal joint. After the animal reached the hospital and the wounds were probed it was decided to take an X-ray picture, Plate 2. This picture, as can be seen, shows in addition to the open joint a simple fracture of the articular surface of the small metatarsal bone, merely con-

firming the clinical diagnosis. In addition, however, it was discovered that there existed a fracture of the small metatarsal bone (splint), about one and one-half inches below the wound on the external surface of the hock. How the fracture of the small metatarsal bone occurred is a question, as there was no mark or other evidence on the skin over the fracture. Presumably it happened by indirect violence, as the upper portion of the splint bone is not as a rule completely ankylosed, as there is an artery (metatarsea dorsalis) interposed between the head of the splint bone and the cuboid, while the lower portion is more or less firmly attached to the large metatarsal bone. This is accomplished by a strong interosseous ligament. Consequently when the blow (kick) occurred it probably forced the upper end or head of the small metatarsal bone forcibly into the cuboid in a direction slightly forward and inward while the lower portion remained stationary with the natural result of a fracture in the middle third of the splint bone, where it is completely ankylosed with the large metatarsal bone.

The animal from the beginning manifested a severe lameness with the tarsal joint considerably swollen. The leg was showered with cold water and a bichloride pack 1:1000 placed on the wound and held in place by a bandage. After a few days of this treatment, the swelling of the hock seemed to be going down a trifle. The escape of synovial fluid from the outside wound would convince the most skeptical that the joint cavity was actually opened. In addition to the above treatment of showering and application packs, an injection of equal parts of Lugol's solution and glycerine was made twice daily. This treatment was inaugurated December 8, and continued until the 14th, when the animal appeared better and stood very well on the leg, bearing equal weight, presumably, on the heel as well as the toe. Up until this time the animal hesitated to place the heel on the ground in standing or walking. On the 15th the animal was noticed in standing to lift the right front leg, consequently placing considerable weight on the affected right hind leg.

The treatment with equal parts of Lugol's and glycerine was continued until December 19th, with the showering and bichloride packs discontinued. The animal at this time showed considerable improvement, and used the leg very well, particularly in walking, where he manifested very little lameness. The wound on the external surface of the hock was entirely healed at this time. The wound on the posterior surface of the hock apparently became healed a few days before. The animal was given complete rest from December 20, 1924, to January 9, 1925. On the latter date a slight discharge was noted coming from the wound on the posterior surface of the hock. The animal also manifested a severe lameness which came on very suddenly when you consider how well he seemed to be improving, prior to this date. A second X-ray picture was taken on this date, Plate 3. This picture shows a small piece of bone, about three-eighths inch in diameter, separated in the tissues. This piece of bone was removed surgically,

January 9, 1925. From this date until January 20, 1925, the cavity was washed out daily with a one per cent solution of sterilac. The wound healed and for several days iodex was applied. The animal was removed from the hospital to the R. O. T. C. stables January 21, 1925. From the latter date until February 10, instructions were given to have the animal exercised by walking for about a half mile morning and evening. After the latter date the animal was turned into the corral and permitted to take voluntary exercise. The leg at this time was apparently getting stronger with no manifestation of lameness on a walk and with only a slight lameness on a trot over a level surface. Lameness is more pronounced on a trot going up grade.

On February 20, the third, and last, picture was taken, Plate 4. This picture shows that healing had taken place in both places where fractures had occurred. February 22, the animal was practically normal. Advised slow work under the saddle, that is, gradual exercise by trotting and slow canter over level road. The animal was dropped from sick report February 27, and marked "Duty Improved." It is confidently expected that with the gradual exercise advised complete recovery will take place in the course of several weeks.

The most interesting phase of this case, in the writer's opinion, is the pictures. Certainly no one would even have suspected, let alone diagnosed, a case of fracture in the body of the splint bone. Then again, it probably would have been a considerable length of time until the free portion of the articular head of the splint bone would have been discovered in its position (loose) in the tissues. As it was, as soon as the pronounced lameness became manifest, an X-ray picture was taken and immediately developed which showed the portion of the splint bone in its position in the soft structure. As soon as it was removed the pronounced lameness disappeared, and the animal made a gradual recovery. On March 12th, instructions were given to have the animal placed in regular work as he is considered serviceably sound, with no manifestation of lameness.

ACKNOWLEDGMENT

Credit is due Prof. Frost and Dr. Mabey, under whose direction the animal was treated, and to Professors Milks and Frost for the taking and developing of the pictures.

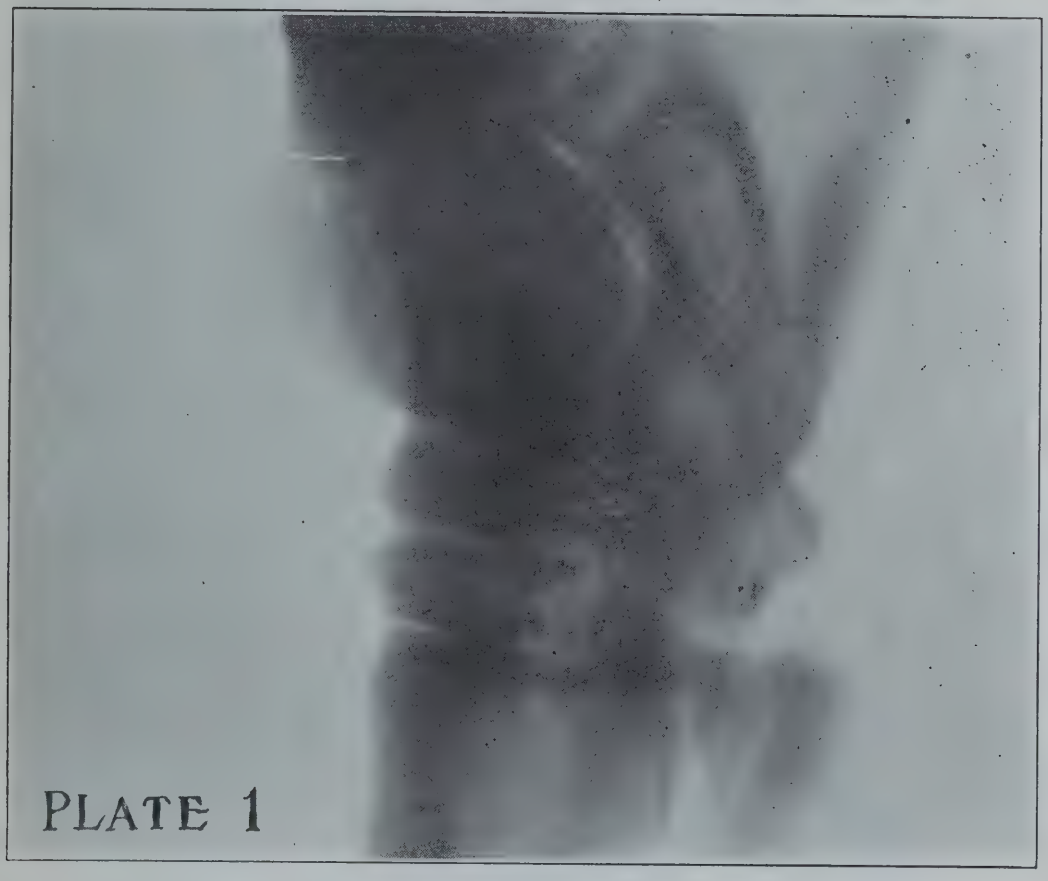


PLATE 1

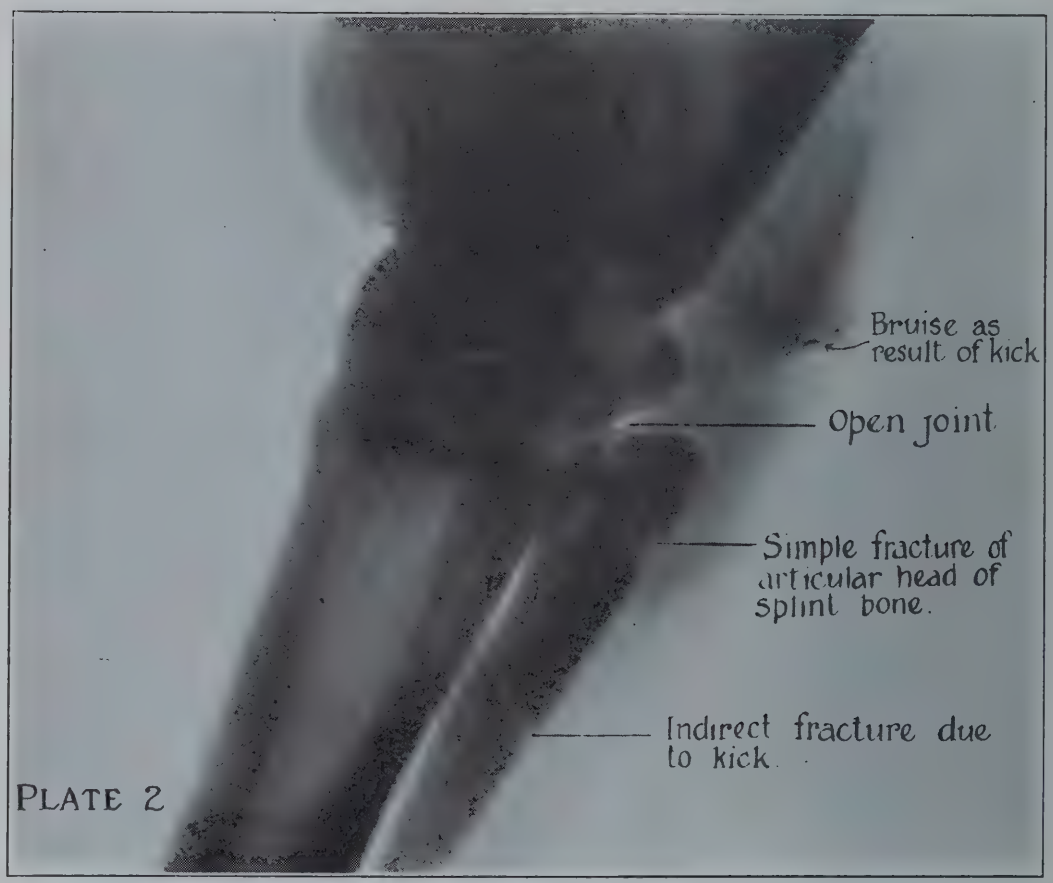

Bruise as result of kick
Open joint
Simple fracture of articular head of splint bone.
Indirect fracture due to kick.

PLATE 2

PLATE 3

PLATE 4

FOWL PLAGUE

E. L. BRUNETT and S. KONDO
Department of Pathology and Bacteriology

The earliest records that we have concerning fowl plague are from Italian investigators. In Italy the ravages of the disease are said to have reached large proportions during the latter part of the 19th century. It remained undifferentiated from fowl cholera until 1880 when Rivolta and Delprato (39) pointed out clinical differences between the two. The name "exudative typhus of birds" was given to the disease by them. During the next twenty years, outbreaks occurred throughout the poultry sections of Italy. A number of investigators reported both as to its occurrence and nature.

The etiology of the disease does not seem to have been determined until 1900. Centanni and Savonuzzi (5), investigating an outbreak in Italy, demonstrated that the etiological factor was not arrested by a Berkefeld filter. Later it was diagnosed in Austria, Germany and Hungary. It is supposed to have been introduced into Germany during the Brunswick Poultry Exhibition, as it appeared in a number of localities immediately afterward.

The following table gives the year in which fowl plague was recognized as a distinct disease of poultry in different countries. It is not assumed that this list is complete or that countries not mentioned are free from it.

Country	*Year first reported*
Italy (39)	1880
Austria (26)	1900
Germany (26)	1901
Hungary (10)	1903
Belgium (26)	1903
France (10)	1904
China (10)	1909
Holland (1) reported in ducks	1920
Holland (1) reported in chickens	1924
United States (2)	1924
Japan (16)	1925

THE OCCURRENCE OF FOWL PLAGUE IN NEW YORK STATE

The presence of the disease in this country was first reported in November, 1924, by this College. Twenty-two outbreaks were recorded by this laboratory from October, 1924, to June, 1925. Sick or dead fowls were received from each outbreak, which enabled us to make an accurate diagnosis. A number of other outbreaks in this State were reported but as material from them was not received, they have not been included.

Krohn (19) estimates the loss of fowls in greater New York to be from 500,000 to 600,000. The losses on the farms and upstate markets can be estimated from the information received at 60,000.

The outbreaks of fowl plague diagnosed in this laboratory and the months in which it occurred are given below.

Year	*Month*	*Number of outbreaks*
1924	October	1
1924	November	3
1924	December	4
1925	January	6
1925	February	2
1925	March	4
1925	April	1
1925	May	0
1925	June	1

It was not possible to obtain a complete history of each outbreak. Some interesting data concerning a few of the flocks follow.

Flock No. 1. The owner purchased a poultry farm with 350 chickens. Four hundred more were added to the flock. The newly purchased fowls were obtained from native flocks and had not been in contact with any markets as far as we were able to determine. He sold about 100 to a New York City dealer who brought crates upon the premises. Thirty-eight days after this incident, the chickens began dying.

The mortality record, carefully kept by the owner, shows the rapid progress the disease made in the flock.

Mortality Record Flock 1.

MONTH	October												
Day	1–15	15	16	17	18	19	20	21	22	23	24	25	26
Mortality	1	1	1	2	11	3	10	15	14	37	50	62	96

MONTH	October					November				
Day	27	28	29	30	31	1	2	3	4	5
Mortality	70	46	58	48	..	54	40	4	4	0

Size of flock October 15	651
Size of flock November 6	24
Total lost in 21 days	627

A neighbor whose hen house was approximately ten feet away from the house of flock 1, did not lose any chickens from fowl plague. The houses faced in opposite directions and the chickens were fenced in on both places. No direct contact between the two flocks was possible. Although no accurate observations were made concerning the wandering habits of a number of cats which lived on the infected premises, the disease apparently was not disseminated by them. This does not exclude the possibility of mammals acting as mechanical carriers.

Flock No. 2. A flock of 1,200 chickens was maintained on this farm. One hundred and two cockerels were purchased from one of the New York City and local markets, to be fattened and later remarketed. These were placed in a pen at a distance from the large hen house with a yard fence between the two. Seventeen days after the cockerels were brought on the place, they began dying. The mortality was 50 per cent when the disease was diagnosed.

The survivors were slaughtered and orders given to disinfect the exposed pen and equipment. It was noticed on this visit to the place that some of the cockerels showing symptoms were allowed about the yard. The fencing around the large laying house prevented their coming in contact with the laying stock.

About two weeks after the disposal of the surviving cockerels, the disease appeared in the laying flock with a loss of 1000, 200 surviving. The carrying of the virus by the owner seemed obvious in this case.

Flock No. 3. A flock of 250 chickens was maintained on this farm. The owner bought some sick chickens for immediate consumption at a bargain price. These were obtained from a chicken dealer who had recently lost a large number of fowls in his market. After preparing the chickens for table use, the offal was thrown to the flock. This was followed by an outbreak of the disease in which 246 chickens died within one week.

Flock No. 4. The owner of a small flock was accustomed to purchase chickens for eating purposes from the New York markets. He also was inclined to retain and add to his flock the laying hens which might be among them. Such recently purchased hens caused the loss of his entire flock.

Flock No. 5. The owner bought some turkeys from outside the state. Three days after adding the purchased turkeys to his flock of turkeys and chickens, the recent additions died. These deaths were followed by the loss of all of his native turkeys and nearly all of his chickens.

Flock No. 6. A turkey was purchased from a market and added to a flock of chickens. The death of the turkey was followed by the loss of nearly the entire flock.

Flock No. 7. A butcher purchased 600 fowls from another dealer; these were added to 300 fowls that were being held in reserve for the holiday season. The loss of nearly the entire lot

resulted. Specimens received from this outbreak had been frozen ten days or more. No difficulty was encountered in obtaining the virus from the carcasses.

Although it was not possible to determine just how the disease was introduced in the flocks in all instances, the majority of outbreaks resulted from the addition of recently purchased poultry.

NATURE OF THE DISEASE.

Symptoms. These are not pathognomonic so that the disease can be differentiated from other acute infections. The affected individual may appear normal and die suddenly without any manifestation of symptoms. In the majority of instances, however, some symptoms are present. Their sudden onset is striking. The affected fowl takes to the roost or some other place where it will not be disturbed. It has a stupid appearance. The feathers may become ruffled, the head and neck bent downward on the breast; the head seems to be raised only with difficulty. In some instances edematous swellings of the head and wattles have been noted. These swellings consist of the exudation of serum into the subcutaneous tissue around the lower part of the head, including the wattles and extending sometimes partially down the neck. This condition, from the observations in this laboratory, seems to be confined to the heavier breeds of chickens. In the experimental work, white leghorn chickens were used largely and the absence of this edematous condition was noted throughout the entire work. In the few instances when specimens of the heavier meat breeds were used, the edematous condition was noted.

Infected chickens sometimes emit a peculiar hiccough sound. Diarrhea was not noted in the natural outbreaks although the experimentally infected chickens invariably showed a profuse watery diarrhea. There may be a mucous discharge from the mouth or nostrils. There is often difficulty in breathing. Food and water are refused and the chicken may stagger or fall if made to move. The beak may open and shut convulsively. The temperature which is usually high at first (110–112° F.) gradually becomes lower (103–100° F.) than normal (106° F.). A comatose condition finally sets in and death usually takes place within twenty-four hours from the commencement of the symptoms.

In the artificial production of the disease, the chickens die in from 36—48 hours after exposure.

Anatomical Changes. Fowl plague cannot always be diagnosed from the lesions alone. The lesions, however, are somewhat characteristic and can be relied upon to a certain extent. The general evidence of septicemia prevails. The principal lesion is the petechiation of the fat tissue which varies in intensity and location.

In the peracute form, lesions are not usually observed. In the acute form a hemorrhagic and an exudative type of lesions are found.

Acute hemorrhagic type. The usual manner of autopsying a fowl consists of first opening up the abdominal cavity. It is here that the lesions may first be seen. The fat tissue forming the floor of the body cavity may be sprinkled with petechiae. The hemorrhages may be found scattered over the fat tissue located in various places around the abdominal cavity. The fat tissue in the coronary groove of the heart is one of the most consistent locations of these hemorrhages. The hemorrhages are rarely found in the cardiac muscles.

Examination of the mucous surfaces reveals an almost constant hemorrhagic or congested condition of the nasal chambers. Invariably a large amount of mucoid material is present. The proventriculus usually shows ill defined hemorrhages, at the transition with the gizzard mucosa. This is considered to be of considerable diagnostic value.

Blood extravasations may be found beneath the tough gizzard lining. The intestines usually do not show marked changes. The absence of a hemorrhagic enteritis usually present in fowl cholera may be used to some extent in eliminating that disease. The esophagus, mouth cavity and trachea may be sprinkled with petechiae. The liver, spleen, kidneys and lungs do not show marked changes. Congestion of these organs, especially the first three mentioned, may be noticeable. The heart blood may or may not be coagulated.

Acute exudative type. The lesions found in this type of the disease consist chiefly of exudations of serum into various tissues and cavities with or without hemorrhages.

Examination of a fowl dead with this particular type of the disease may show a swollen condition of the head and neck. Incisions made into the subcutaneous tissue will reveal a clear straw colored fluid, gelatinous in consistency. This condition seems to be confined to the heavy meat breeds as previously mentioned.

The abdominal cavity contains varying amounts of a clear, straw colored coagulum. Petechiae may be present over the various fat tissues.

The lungs are congested and may be infiltrated with extravasated serum. A layer of clear coagulated serum may be found to have separated the diaphragm from the lung tissue. The liver, spleen and kidneys do not show marked changes. Hemorrhages may be present in the trachea, esophagus and nasal chambers. The large amount of mucoid material in the nasal passages is noticeable.

The following table summarizes the autopsy findings in 50 artificially infected chickens.

AUTOPSY FINDINGS.

Hemorrhages Mucous Surfaces	Number	Per cent
Nasal chambers	32	64
Proventriculus	27	54
Trachea	23	46
Gizzard, beneath lining	20	40
Esophagus	17	34
Intestine	3	6
Serous Surfaces		
Abdominal cavity	44	88
Epicardium	43	86
Pericardium	39	78
Proventriculus	21	42
Gizzard	6	12

Exudates.

	Serous		Mucus	
	Number	Per cent	Number	Per cent
Nasal chambers	....	....	42	84
Thoracic cavity	18	36	....	....
Abdominal cavity	15	30	....	....
Subcutaneous (wattles, head, neck)	....	....	....	....
	Coagulated		Uncoagulated	
Heart Blood	21	42	29	58

Organs

	Congested		Discolored		Edema		Normal	
Lungs	14	28%	....	....	18	36%	18	36%
Spleen	....	....	....	....	....	....	50	100%
Kidneys	12	24%	....	....	....	....	38	76%
Liver	17	34%	16	32%	....	....	17	34%

NATURE OF THE ETIOLOGICAL FACTOR OF THE DISEASE.

1. *Filterability of the virus*

Since Centanni and Savanuzzi (5) reported in 1900 that the virus of fowl plague was not arrested by the Berkefeld filter, attempts have been made to determine the arresting properties of various other filters.

Lode (22) in 1902 reports variable results in his attempts to pass the virus through Chamberland, Pukall, Hauser and Kitasto filters. These results were confirmed by Centanni (4) and also by Ostertag and Wolffhügel (36).

Depperich (6) reports that he was successful in passing the virus through the Berkefeld-Nordtmeyer filter but not successful when the Chamberland filter was used.

Maggiora and Valenti (27) report that the Chamberland filter 'K' did not arrest the virus whereas the Chamberland filter 'F' arrested it.

Calmida (3) reports the passage of the virus through the Chamland filter 'B'.

Rosenthal (40) states that the virus is not retained by the Pukall and common porcelain filters whereas the Reichel filter retains the virus.

Giemsa and Prowazek (7) state that the virus does not pass through the Pukall filter which has been covered with an agar solution.

Hertel (9) was able to pass the virus through the Berkefeld, Chamberland B & F, Pukall and Porcelaine d'Amiante filters. Using the Reichel and Paul Altmann (Berkefeld) filters he obtained variable results.

No attempts were made in the course of this work to determine the arresting qualities of the different types of filters.

The Berkefeld 'N' filter was used whenever filtration was desired. The technique consisted of suspending whole blood or ground tissue extract from suspected material in approximately fifty times its volume of sterile saline. The coarse particles were removed by filtering through cheese cloth. A sufficient amount of a fresh bouillon culture of *P. aviseptica* was added to the suspension; the material was then passed through a Berkefeld 'N' filter using a small faucet pump for suction. Rabbits inoculated with 0.25 mil doses of the material before filtration, died from avisepticus septicemia. The inoculation of 5 mils of the filtrate into rabbits failed to produce any ill effects.

Chickens inoculated with varying amounts of the filtrate died promptly showing typical lesions of the disease when the virus was present. The inability to infect rabbits with the large doses of the filtrate demonstrated the efficacy of the filters used.

2. *Susceptibility of various species*

The question of susceptibility is one which has been given some attention by numerous investigators. A search of the literature reveals some interesting reports.

Centanni (5) reports the turkey, goose and guinea fowl susceptible to the virus. The inoculation of rabbits gave variable results. Young pigeons were found by him to be susceptible whereas old pigeons were not. The attempts to produce symptoms or fatal results with the virus in 2 guinea pigs, 1 rat, 1 dog, and 1 fox were negative.

Scheurlen and Buhl (42) could not produce the disease in mice, rabbits, guinea pigs, pigeons and ducks.

Greve (8) was able to produce the disease in sparrows and bantam chickens but not in pigeons and mice.

Lüpke (24) states that the rabbit, mouse and pigeon are not susceptible but sparrows are.

Maggiora and Valenti (28) report that they were able to produce the disease by both feeding and subcutaneous inoculation of the virus in sparrows, starlings, gold finches, owls and falcons. Rabbits, guinea pigs, mice and pigeons were found not to be susceptible.

Joest (13) reports his inability to infect pigeons, mice and guinea pigs.

Lode and Grüber (23) were unable to produce the disease in guinea pigs, mice and rabbits. Their results with pigeons were variable.

Hertel (9) obtained negative results with geese, ducks, pigeons, rabbits, mice and guinea pigs, in his attempt to infect them. The sparrow was found to be susceptible.

Ostertag and Wolffhügel (37) state that sparrows and turkeys are susceptible to the disease. Ducks, mice, guinea pigs, swine and rabbits according to the same investigators are not susceptible. Young geese were found by them to be susceptible.

Kleine and Moeller (15) also state that young geese are susceptible.

Kraus and Schiffmann (18) report that young geese are unable to withstand intramuscular injections of the virus and subdural injections of the virus into old geese will produce fatal results.

Kraus and Doerr (17) have also reported similar findings.

Krumwiede, Gerber and Provost (20) found the pigeons, turkey and guinea hen to be susceptible. The subcutaneous inoculation of ducks and geese gave variable results. Mice, guinea pigs, rabbits, and monkeys (Macacus) were found not to be susceptible.

Stubbs (43) inoculated 16 pigeons subcutaneously with the virus with two resulting deaths. He was able to demonstrate the virus in the brain tissue of one of them.

In this laboratory there has been occasion to test the susceptibility of the most frequently used experimental animals, the rabbit, guinea pig and white mouse.

Rabbits remained unaffected from the subcutaneous inoculation of 5 mils of the filtered virus blood. The inoculation of virus liver and spleen also gave negative results. Intraperitoneal inoculations of similar material were not fatal to rabbits.

Guinea pigs inoculated intraperitoneally with 1 mil doses of a virus liver suspension remained unaffected.

White mice were not affected by the subcutaneous inoculation of virus liver extract in 0.25 mil doses.

Five sparrows were obtained by trapping.

Three small pieces of bread were soaked in virus blood obtained from an artificially infected chicken. The sparrows consumed the bread in one hour's time. One sparrow died three days later, another died four days later, the third died five days later and the remaining two were dead on the seventh day after being exposed.

Five to seven hours before death, general depression was noticed in all of the sparrows. The lack of control over the legs, droopiness and inability to fly to the roost were the outstanding symptoms.

The sparrows were examined bacteriologically and adult chickens were inoculated separately with material from each one. The bacteriological examination was negative in all cases. The inoculum used to inject the adult chickens was prepared by grinding up a small portion of the sparrow's liver and suspending it in saline. The subcutaneous inoculation of 0.2 mil of the suspension produced fatal results in all of the chickens in 1.5 days. The lesions and bacteriological examination of the inoculated chickens demonstrated the presence of the virus in all the livers from all of the sparrows.

The determination of the susceptibility of pigeons and ducks was next attempted.

Twenty adult pigeons, sixteen adult ducks and twenty-six adult chickens were used. Five different methods of exposure were used. The virus used consisted of the whole blood diluted 1–10 with saline containing sufficient potassium citrate to prevent coagulation.

The table following summarizes the experiment. All the fowls were exposed to the same virus at the same time. They were kept under observation for 5 weeks.

The technique of exposure by feeding the virus consisted in pipetting it directly down the esophagus. The subcutaneous inoculations were made over the breast muscles. The method employed in exposing the fowls through the conjunctival sac consisted of dropping the virus directly into the sac and holding the eyelid closed until the inoculum was distributed well around the cavity. In the fourth method the virulent blood was placed in the nasal chambers through the nostril by means of a pipette. In placing the virus in the trachea through the larynx, care was taken not to allow the mucous membranes of the mouth to become contaminated with the virus.

TABLE

Feeding	Subcutaneous inoculation	Conjunctival sac	Nasal chambers	Trachea
6 chickens	2 chickens	6 chickens	6 chickens	6 chickens
1 mil blood	0.5 mil blood	1 gtts. blood	4 gtts. blood	3 gtts. blood
Positive 5	Positive 2	Positive 5	Positive 3	Positive 5
Negative 1	Negative 0	Negative 1	Negative 3	Negative 1
4 pigeons	4 pigeons	4 pigeons	4 pigeons	4 pigeons
0.2 mil blood	0.25 mil blood	1 gtt. blood	2 gtts. blood	3 gtts. blood
Positive 0	Positive 0	Positive 0	Positive 0	Positive 0
Negative 4	Negative 4	Negative 4	Negative 4	Negative 4
4 ducks	4 ducks	4 ducks		4 ducks
1 mil blood	1 mil blood	2 gtts. blood		6 gtts. blood
Positive 0	Positive 0	Positive 0		Positive 0
Negative 4	Negative 4	Negative 4		Negative 4

Key — Positive died.
Negative remained alive.

The surviving chickens were inoculated subcutaneously five weeks after the primary exposure. All succumbed to the disease. The inability to produce symptoms or fatal results in the adult pigeons and adult ducks points toward their non-susceptibility. The virulence of the virus is shown by its effect on the chickens.

Five weeks after the primary inoculation of the four ducks they were reinoculated subcutaneously with the virus blood from chickens. Sufficient control chickens were used to determine that the blood was virulent for chickens at the time it was used.

DUCKS

	No. 1	No. 2	No. 3	No. 4
Primary injection:				
Dose	1 mil	1 mil	1 mil	1 mil
Second injection:				
Number of days after first injection	35	35	35	35
Dose	1 mil	1 mil	1 mil	1 mil
Third injection:				
Days after second injection	5	5	2	2
Dose	5 mils	5 mils	1 mil	1 mil
Fourth injection:				
Days after third injection	16	16	3	3
Dose	15 mils	10 mils	3 mils	3 mils
Fifth injection:				
Days after fourth injection	.....	.....	4	4
Dose	.....	.....	5 mils	5 mils
Sixth injection:				
Days after fifth injection	.....	.....	6	6
Dose	.....	.....	10 mils	10 mils

The ability of these four ducks to withstand repeated doses of the virus blood is evidenced by the table. The ducks were sacrificed two weeks after the last injection and the sera collected for use in another experiment.

The susceptibility of the turkey was demonstrated during the year. A poultry man received several turkeys from another state and allowed them to mingle with his small flock of chickens. Soon after receiving them, the turkeys died. Their death was followed by the loss of nearly the entire flock. Filtration experiments with tissue extracts from one of the dead turkeys together with post mortem findings and bacteriological examination, demonstrated the presence of the virus in the turkey. Adult chickens were readily killed with the virus obtained from the turkey. The chickens from the same flock gave similar results.

INFECTING DOSE OF THE VIRUS BLOOD

Maue (33) reports being able to infect chickens with the virus blood diluted 1–100,000 times. He was not successful with a dilution of 1–1,000,000.

Maggiora and Valenti (29) were able to produce the disease with 4 mils of a 1–125,000,000 dilution of the virus blood.

Landsteiner (21) separated the cellular elements of the virus blood from the serum by centrifugation and found that the serum was infectious in 0.5 mil doses when diluted 1–100,000. In dilutions of 1–10,000,000 the serum in 0.5 mil doses was innocuous. The cellular elements of the blood when diluted 1–1,000,000 were infectious in 0.5 mil doses. Diluting the cellular elements 1–10,000,000 and 1–100,000,000 gave variable results.

Kitt (14) reports the minimum infective dose as 1 mil of the virus blood diluted 1–1,000,000.

Kleine and Moeller (15) report that the virus blood from geese is not as virulent as that from the chicken. They were able to infect chickens with 2 mils of the virus goose blood but a dose of 0.2 mil of the same blood gave negative results.

The lethal dose of one of the viruses in use in this laboratory was determined after successive passages of it through then adult chickens in a period of about one month. One mil of the virus blood diluted 1–100,000,000, inoculated subcutaneously into chickens, produced fatal results. Later in the year when the virus was being inoculated only once monthly a decrease in its virulence was noted. The appended table shows the increase in the infective dose or virulence of the virus.

The virus was passed successively through five adult chickens from October 29 to November 12 and then tested as indicated below. The serial inoculations were continued on five more adult chickens until December 4, when the virus was tested again. The decrease in the lethal dose is shown in the table. The subcutaneous introduction of the virus was as effective as the intravenous method.

INCREASE IN VIRULENCE

DILUTION	OCTOBER 29–NOVEMBER 12, 5 fowls	NOVEMBER 12–DECEMBER 2 5 fowls	
	1 mil intravenously	1 mil intravenously	1 mil subcutaneously
1–100	Positive	.....	.....
1–1,000	.....	.....	Positive
1–10,000	Positive	.....	Positive
1–100,000	.....	Positive	Positive
1–1,000,000	Negative	Positive	Positive
1–10,000,000	.....	Positive	Positive
1–100,000,000	Negative	.....	Positive
1–10,000,000,000	Negative	.....	.....

Key — Positive died.
Negative lived.

THERMAL RESISTANCE OF THE VIRUS

The literature contains much information regarding the thermal resistance of the virus.

Lode and Grüber (23) found that a suspension of the liver from an infected fowl retained its virulence when heated 30 minutes at 60°C.

Centanni (4) states that heating the virus for 60 minutes at 57–58°C will not destroy its virulence. Exposing to a temperature of 60°C for 60 minutes will render it avirulent.

Hertel (9) reports that a suspension of the virus liver in saline

is rendered innocuous when heated for 30 minutes at 60° C but is not affected by a temperature of 50°C for the same length of time.

Maue (33) states that the virus is destroyed in blood by heating at 60°C for 30 minutes but is not destroyed when heated at the same temperature for 20 minutes.

Freezing temperature apparently does not destroy the virus. During the course of the year we received specimens (unopened carcasses) which were frozen and had been for at least 10 days. No difficulty was experienced in reproducing the disease by inoculating portions of the liver, spleen and heart blood.

In our experiments we used the "A" virus which was obtained from an artificially infected chicken which had died of the disease. A small portion of the heart blood was collected and added to an equal portion of physiological salt solution which prevented coagulation. In one-half of the experiment sodium citrate solution (0.9%) was substituted for saline.

The diluted virus was drawn into capillary glass tubes which were hermetically sealed after filling. The capillary tubes were approximately 0.5 mm. thick, 4 to 6 cm. long, tapering to a point at both ends, and from 0.5 to 0.75 cm. in diameter at the center.

A simple water bath was used. The water was heated to the desired temperature, which was maintained by the frequent application of heat. The glass tubes containing the virus blood were placed in the water bath after the water had reached the proper temperature.

At the end of the different periods, the tubes were removed and immediately placed in cold water. In no instance was the blood found to have coagulated. Inoculations were made soon after cooling by injecting intravenously 0.2 mil of the heated virus blood into normal adult chickens, one chicken being used in each case. The examination of the chickens that died showed lesions characteristic of the disease. In the two instances where the virus blood failed to produce death, no symptoms were observed in the inoculated chickens. Bacteriological examinations were made in each case and they were uniformly negative. The chickens that survived in two instances were later tested and found not to be immune.

Results are summarized in the appended table.

Temperature	Heating period	Result
45° C	30 minutes	*
50° C	30 minutes	*
55° C	30 minutes	*
55° C	15 minutes	*
60° C	30 minutes	†
60° C	5 minutes	*
65° C	1 minute	†
Unheated	.....	*

Key — * Chicken died.
† Chicken not affected.

LONGEVITY OF THE VIRUS

Centanni (4) was able to keep the virus blood virulent in hermetically sealed tubes for 3 weeks in the summer and for 3 months during the spring weather. The multiplication of putrefying organisms in material containing the virus results in its attenuation or destruction in 3 days. Slices of the heart and lungs containing the virus retained their virulence for 30 days when kept in glycerin.

Lode and Grüber (23) report the ability of the virus to retain its virulence in the cadaver for 33 days but not for 37 days when kept at 0°C. They also state that putrefaction destroyed the virus in a cadaver in a few days. The virus in the droppings was also destroyed in a few days by putrefaction.

Maggiora and Valenti (30) were able to keep the virus blood 42 days.

Kitt (14) obtained variable results with the virus brain soaked in glycerin.

Giemsa and Prowazek (7) were able to store the virus in 30–40 per cent glycerin at ice box temperature for 5 months.

Ostertag and Bugge (35) report the retention of the virulence of virus blood kept in capillary tubes at 5°C for 83 days.

Maue (33) reports that he was able to keep the virus blood virulent for 144 days when kept at 8°C. The virus blood diluted with equal parts of glycerin and kept at 8°C remained virulent at 270 days.

Throughout the course of this work no difficulty was encountered in storing the virus at ice box temperature. The virus retained its virulence for 45 days in unopened carcasses. Whole virus blood kept in hermetically sealed glass tubes at ice box temperature retained the virulence for 2 months.

The following table gives the viability of the virus with glycerin at two different temperatures. Equal parts of the virus tissue and pure glycerin were used in all cases. Where three constituents made up the preparation, equal parts of all three were used. The tissue was thoroughly ground up before suspending in glycerin. Ox bile was used in two of the preparations.

In the last preparation, normal chicken's liver was used. The preparations were kept in hermetically sealed glass tubes and bacterial multiplication was not detected in any of the samples.

At varying periods, adult chickens were inoculated subcutaneously.

	Icebox 8° C Virulence		Incubator 37.5° C Virulence	
PREPARATION	Retained	Destroyed	Retained	Destroyed
Brain and glycerin	100 days	.....	36 hours	48 hours
Blood and glycerin	78 days	.....	84 hours	96 hours
Spleen and glycerin	70 days	.....	.....	12 hours
Blood, bile, glycerin	70 days	.....	48 hours	.....
Blood, bile	30 days	50 days	.....	12 hours
Liver, glycerin	30 days	45 days	12 hours	24 hours
Blood, n. liver, glycerin	30 days	50 days	12 hours	24 hours

The longevity of the virus at 8° C in the first four preparations has not been completed but the viability is apparently marked. The data presented by the last four preparations are interesting. It was noted that a suspension of the virus liver in glycerin was not extremely viable, as indicated in the table. The addition of normal chicken liver to virus blood and glycerin gave similar results. The bile content of the liver tissue exerts an attenuating effect upon the virus. Ox bile added to virus blood attenuated it whereas the addition of glycerin to a similar preparation decreased the rapid attenuation.

In the attempt to produce an immunizing agent, further information was obtained concerning the viability of the virus.

	37.5° C Virulence		20–23° C Virulence	
PREPARATION	Retained	Destroyed	Retained	Destroyed
Liver, phen. saline 1–5	.....	24 hours	.....	.....
Liver, glyc. saline 1–5	24 hours	48 hours	.....	.....
Liver, phen. gly. sal. 1–5	.....	24 hours	.....	24 hours
Spleen, phen. glyc. sal. 1–5	.....	.....	20 days	23 days
Brain, phen. glyc. sal. 1–5	5 days	7 days	32 days	35 days
Brain, phen. sal. 1–50	55 days	65 days	.....	.....

The 1–5 refers to the proportion of the virus organ to the suspending medium. The phenolized saline consists of 0.5 per cent phenol in physiological salt solution. Glycerin saline consists of equal parts of glycerin and physiological salt solution. Phenolized glycerin saline was made up on glycerin saline plus 0.5 per cent phenol. The viability of the spleen and brain tissue is marked. The extreme viability of the virus brain suspended in phenolized saline is striking.

TRANSMISSION OF THE VIRUS

Lüpke (25) and also Jess (11) reproduced the disease readily in chickens by feeding the nasal mucus and organs from diseased fowls.

Joest (12) found the intestinal contents, organs and blood infective either by feeding or subcutaneous inoculation.

Lode and Grüber (23) were able to demonstrate the virus in the blood, organs, intestinal contents and bile by feeding, subcutaneous or intramuscular exposure.

Centanni (4) besides confirming the presence of the virus in the blood organs and droppings examined blood sucking mites with negative results.

Hertel (9) produced the disease by placing virus spleen extract in the conjunctival sac and pharynx. The contamination of the nostril with virus blood and the feeding of intestinal contents also gave positive results. He was able to reproduce the disease through the use of lice.

Ostertag and Wolffhügel (36) reproduced the disease from the heart blood, liver, spleen and kidney, by subcutaneous, intramuscular, intravenous and intraperitoneal inoculations. Feeding also

of material from the same sources and also intestinal contents readily produced the disease. The contamination of the nostrils of healthy chickens with the nasal mucus from the diseased fowls, proved fatal in two out of six trials. Attempts to show the airborne possibilities of the virus were negative.

Marchous (32) states that a special ascaris is capable of transmitting the disease and that the fowl tick (*Argus persicus*) is not concerned in the dissemination.

The inoculation of virus blood, spleen, liver and brain, subcutaneously, intravenously and intramuscularly proved fatal in all instances. Feeding of the above mentioned tissues produced the disease in chickens.

The pipetting of 1 mil of the virus blood into the mouth of six adult chickens resulted in the death of five. One drop of virus blood placed in the conjunctival sac of six adult chickens proved fatal to five. Four drops of virus blood placed in the nasal chamber of each of six adult chickens produced the disease in three. Three drops of virus blood placed in the trachea of each of six adult chickens resulted fatally for five of them. The survivors were unable to resist the virus introduced subcutaneously later.

The nasal mucus, droppings and the blood drawn from the wing vein of artificially infected chickens at the onset of symptoms proved infective.

The viability of the virus in the nasal mucus, blood and intestinal contents depends upon the degree at which bacterial multiplication progresses. The virus was readily found in the intestinal contents, nasal mucus and blood from carcasses exposed to summer temperature several hours.

Attempts were made to infect chickens by placing them in cages contaminated by artificially infected chickens. After removing the carcasses of the dead chickens, normal chickens were placed in the cages without changing the litter or feed cups. Normal chickens were also placed in the same cages with inoculated chickens and kept there after the inoculated ones had died and been removed. In all instances the normal chickens failed to show signs of infection. They were all inoculated later and found to be susceptible.

The rapid destruction of the virus by bacterial action seems to play an important part in this disease. The fact that the disease did not spread extensively may possibly be accounted for in this manner.

REFERENCES

1. BARION. Ann. Med. Vet., Vol. II, Nov. 1924. Abst. from Tijdschrift voor Diergeneskunde.
2. BRUNETT. Jour. A. V. M. A., New Series, Vol. 19, No. 4, p. 497, 1925.
3. CALMIDA. Cited by Ostertag, Handb. d. path. Mikroorg. von Kolle und Wassermann. Bd. 6, p. 280, 1913.
4. CENTANNI. Centralbl. f. Bakt. 1 Abt. Orig. Bd. 31, No. 5, p. 182, 1902.
5. CENTANNI AND SAVONUZZI. Centralbl. f. Bakt. 1 Abt. Orig. Bd. 31, No. 4, p. 142, 1902.
6. DEPPERICH. Cited by Ostertag. Handb. d. path. Mikroorg. von Kolle und Wassermann. Bd. 6, p. 280, 1913.

7. GIEMSA AND PROWAZEK. Cited by Ostertag, Handb. d. path. Mikroorg. von Kolle und Wassermann. Bd. 6, p. 280, 1913.
8. GREVE. Cited by Ostertag and Wolffhügel. Monatsch. f. prakt. Tierheilk. Bd. 14, p. 49, 1902.
9. HERTEL. Arb. a. d. Kais. Ges-Amt. Bd. 20, H. 3, p. 453, 1904.
10. HUTRYA AND MAREK. Path. & Therap. of Dom. An., Vol. I.
11. JESS. Cited by Hertel, Arb. a. d. Kais. Ges-Amt. Bd. 20, H3, p. 453, 1904.
12. JOEST. Cited by Hertel, Arb. a. d. Kais. Ges-Amt. Bd. 20 H3, p. 453, 1904.
13. JOEST. Cited by Ostertag and Wolffhügel. Monatsch. f. prakt. Tierheilk. Bd. 14, p. 49, 1902.
14. KITT. Cited by Ostertag. Handb. d. path. Mikroorg. von Kolle und Wassermann. Bd. 6, p. 280, 1913.
15. KLEINE AND MOELLER. Centralbl. f. Bakt. 1 Abt. Orig. Bd. 39, p. 545, 1905.
16. KONDO. Personal communication.
17. KRAUS AND DOERR. Centralbl. f. Bakt. 1 Abt. Orig. Bd. 46, H 8, p. 709, 1908.
18. KRAUS AND SCHIFFMANN. Centralbl. f. Bakt. 1 Abt. Orig. Bd. 43, p. 825, 1907.
19. KROHN. Jour. A. V. M. A. New Series, Vol. 20, No. 2, p. 146, 1925.
20. KRUMWIEDE, GERBER, PROVOST. Jour. A. V. M. A. New Series, Vol. 20, No. 2, p. 171, 1925.
21. LANDSTEINER. Centralbl. f. Bakt. 1 Abt. Ref. Bd. 38, p. 540, 1906.
22. LODE. Centralbl. f. Bakt. 1 Abt. Orig. Bd. 31, No. 5, p. 182, 1902.
23. LODE AND GRÜBER. Centralbl. f. Bakt. 1 Abt. Orig. Bd. 30, No. 16, p. 593, 1901.
24. LÜPKE. Cited by Ostertag and Wolffhügel. Monatsch. f. prakt. Tierheilk. Bd. 14, p. 49, 1902.
25. LÜPKE. Cited by Hertel. Arb. a. d. Kais. Ges-Amt. Bd. 20, H 3, p. 453, 1904.
26. MCFADYEAN. Jour. Comp. Path. & Therap. Vol. 21, p. 168, 1908.
27. MAGGIORA AND VALENTI. Cited by Ostertag and Wolffhügel. Zeitschr. f. Hyg. u. Inf. Bd. 42, p. 185, 1913.
28. MAGGIORA AND VALENTI. Cited by Ostertag and Wolffhügel. Monatsch. f. prakt. Tierheilk. Bd. 14, p. 49, 1902.
29. MAGGIORA AND VALENTI. Cited by Maue. Arb. a. d. Kais. Ges-Amt. Bd. 21, p. 537, 1904.
30. MAGGIORA AND VALENTI. Cited by Hertel. Arb. a. d. Kais. Ges-Amt. Bd. 20, p. 453, 1904.
31. MAGGIORA AND VALENTI. Cited by Kraus. Centralbl. f. Bakt. 1 Abt. Orig. Bd. 43, p. 825, 1907.
32. MARCHOUS. Cited by Ostertag. Handb. path. Mikroorg. von Kolle und Wassermann. Bd. 6, p. 280, 1913.
33. MAÙE. Arb. a. d. Kais. Ges-Amt. Bd. 21, p. 537, 1904.
34. MAUE. Arb. a. d. Kais. Ges-Amt. Bd. 21, p. 539, 1903.
35. OSTERTAG AND BUGGE. Zeitschr. f. Infkt. d. Haustiere. Bd. 2, p. 1, 1906.
36. OSTERTAG AND WOLFFHÜGEL. Monatsch. f. prakt. Tierheilk. Bd. 14, p. 49, 1902.
37. OSTERTAG AND WOLFFHÜGEL. Zeitschr. f. Inf. d. Hausetiere. Bd. 2, p. 1, 1906.
38. PROWAZEK. Cited by Ostertag. Handb. d. path. Mikroorg. von Kolle und Wassermann. Bd. 6, p. 820, 1913.
39. RIVOLTA AND DELPRATO. Book of Ornithology, 1880.
40. ROSENTHAL. Cited by Ostertag. Handb. d. path. Mikroorg. von Kolle und Wassermann. Bd. 6, p. 280, 1913.
41. RUSS. Arch. f. Hyg. Bd. 59, p. 286, 1906.
42. SCHEURLEN AND BUHL. Cited by Ostertag und Wolffhügel. Monatsch. f. prakt. Tierheilk. Bd. 14, p. 49, 1902.
43. STUBBS. Jour. A. V. M. A. New Series. Vol. 20, No. 2, p. 180, 1925.

III. IMMUNIZING YOUNG PIGS AGAINST HOG CHOLERA

J. W. BENNER
Veterinary Experiment Station

An experiment on the problem of immunizing young pigs against hog cholera was performed in 1922 and repeated in 1923 and 1924. The object, plan, execution and results of the work in 1922 and 1923 have been described in Annual Reports of the College published subsequent to each year.[1, 2] The object of the experiment in 1924 remained the same, i. e., to learn whether the simultaneous treatment, also the virus alone treatment, confer a solid and lasting immunity to pigs farrowed by and suckling immune, grain fed mothers when the treatment is administered at three weeks of age. The plan of the work in 1924 was practically the same as in the previous years. The execution of the experiment in 1924 differed principally from that of the preceding two years in the method of giving the trial injection of virus to test the immunity.

The results in 1924 differ strikingly from those of 1922 and 1923, especially with the animals that received a trial injection in a radically different manner. The intervals between vaccination and the trial injection in various individuals were about the same as in 1923 but considerably longer than the time allowed in 1922. The dosage of serum and virus used in the simultaneous treatment and also in the virus alone treatment was the same in all three years.

In addition to the foregoing factors and the factor of age of the pigs when vaccinated, there are others which may be found after further study to influence the final conclusions of this work. Among those which may vary in unknown ways are the immunizing properties of the serum and virus used in vaccinating; the grade of immunity of immune sows and the transmissibility of the sow's grade of immunity to the fetus in utero and later through her milk; how long an immune sow will maintain and transmit a high grade of immunity; the concentration of virus contamination in the environment of immune sows and its effects upon the young before and after farrowing.

Such unknown factors involved in the immunizing of young pigs give it all the appearances of a complex and puzzling problem. Further experimentation and study are greatly needed in order to learn the significance of such factors. When this knowledge is obtained the solution of the problem may become simple enough.

TABLE 1—1924 PIG IMMUNITY EXPERIMENT

Litter identification number	Total number farrowed	Dead when farrowed	Died before vaccination	Given simultaneous treatment at three weeks of age (Serum 10 cc., virus, 1 cc.)	Given virus alone treatment at three weeks of age. (Virus, 1 cc.)	Died after vaccination	Conditions observed
1.....	13	9	.....	3	1	.....	
3.....	13	6	.....	6	1	1 virus...	Injury and cholera
5.....	11	3	.....	7	1	.....	
4.....	10	1	.....	8	1	.....	
2.....	10	3	.....	6	1	.....	
7.....	9	4	.....	4	1	1 simult..	Change in feed and cholera
						1 virus...	Change in feed and cholera
9.....	11	6	.....	4	1	1 virus...	Cholera
10.....	7	.....	2	4	1	.....	Cholera-like lesions
11.....	7	.....	7	.....	.....	.....	Sow slow to give milk. Poor suckler. Cholera
12.....	8	1	1*	5	1	2† simult.	1* cholera-like lesions
						1‡ virus..	2† Injury and cholera
6.....	6	2	.....	3	1	.....	1‡ Cholera
							3 Simultaneously treated, crippled and sick. Virus pig O. K.
Totals.	105	35	10	50	10	7	

In the 1924 experiment, out of 70 live pigs farrowed 10 died before vaccination and 7 after vaccination, as shown in Table 1. Of the 10 that died before vaccination, 1 died in 24 hours after birth and showed cholera lesions. Two died in 4 days and showed cholera lesions. Of the 7 that died before vaccination in litter 11, all showed cholera lesions. Of the 7 that died after vaccination, 4 had received virus alone and 3 had had the simultaneous treatment. All showed enough clinical symptoms, or postmortem lesions, or both, to incriminate hog cholera virus as a cause of death. But causes other than cholera virus could also be determined as mentioned in Table 1. Cholera lesions were present so regularly in the pigs that died, both before and after vaccination, that it raised the question whether the pigs were being born with them. Hog cholera lesions in aborted fetuses have been reported but I have not found a record of this condition in pigs that appeared normal when farrowed.

The last sows which we have had farrow, some of which were kept under the same conditions but which have farrowed since Table 1 was made, have thrown some light on this point. It is to be regretted now that little or no attention was given to pigs expelled dead before this time, except that in 1923 our sows farrowed so many dead pigs that an investigation for infectious abortion was made by Dr. Gilman of the Experiment Station staff with negative results.

The following cases are described to give the history of the pigs examined for cholera-like lesions. July 30, 1924, an autopsy was held on one pig that had been laid upon and killed by sow 13 the

second night after his birth. The bladder, kidneys and lymph glands showed almost typical lesions of hog cholera. The mother of this pig was a long-time immune and had been on contaminated ground since conception. On August 3, sow 17 gave birth to 10 pigs, 6 alive and 4 dead. In autopsies on three, cholera-like lesions were found in the bladder, kidneys and lymph glands. The fourth pig was placed in the ice box and on autopsy the next day, August 4, showed practically the same lesions. To aid further in diagnosis I injected 1.5 cc of heart-blood into a susceptible shoat weighing 70 pounds. On the same day one of the pigs from this litter was killed. Hog cholera-like lesions were found and 1.5 cc of the heart-blood of this pig was also injected into a 70-pound shoat to aid in diagnosis.

June 13, I had injected a susceptible pregnant sow with an experimental serum and virus mixture, mixed in the proportion of 20 cc of serum to 0.5 cc of virus. She was kept on cholera-free ground, checked by susceptibles, and on August 2, farrowed 5 pigs alive and apparently well. On August 4, I killed one of these pigs and found practically the same lesions found in the pigs from the long-time-immune sow kept on badly contaminated ground from date of conception. There were no differences found in pigs from immune sows exposed to natural infection and those from susceptible sows simultaneously treated while pregnant. A 70-pound susceptible shoat was injected with 1.5 cc of the heart-blood of the pig from this litter and a susceptible shoat of the same age and size was placed in the pen with the three injected. These four shoats were observed for eight days and no clinical symptoms of cholera were noticed. They were later injected with virus and found to be susceptible.

On May 14, a pregnant sow was injected with 70 cc of the same experimental stock serum and virus mixture. On July 11 she was moved from cholera-free ground to contaminated ground and given 2 cc of potent virus alone to test her immunity. Her temperature was taken daily for one week. No rise occurred. She remained well and on August 5 farrowed 6 pigs apparently normal.

On August 6, a sow that had had the very same treatment as the one just described farrowed 7 pigs, 5 apparently normal and 2 dead. The dead pigs showed cholera lesions. The night after farrowing, one of the five was laid upon and killed. Postmortem lesions in this pig were like those of hog cholera.

Judging from the lesions observed in pigs at birth or shortly after birth, and from the results of the injections of the heart-blood into susceptible shoats, the significance of the lesions in pigs of this age is questionable. However, because of this phenomenon there is some evidence that one should know the history of pigs before birth, as well as afterwards, if it is desired to give them permanent protection from cholera in the most economical way.

Table II gives data on the injection of virus given to the pigs in the experiment of 1924 for the purpose of testing the quality and permanency of their immunity. The individuals of the litters are given in the order in which they received the trial injection of virus.

TABLE II — 1924 PIG IMMUNITY EXPERIMENT

The letter I following the quantity of virus given in the trial injection means intravenously injected; M means intramuscularly

Pig No.	Days from vaccination to trial injection	Virus given in trial injection—cc	Results of trial injection
Litter 1 — Vaccinated March 24, 1924			
3	232	925I	Died 9th day. Hog cholera.
4	232	925I	Remained well.
1	239	2M	Killed 7th day. Hog cholera.
2	250	2M	Died 7th day. Hog cholera.
Litter 3 — Vaccinated May 9, 1924			
4	231	2M	Remained well.
6	231	2M	Remained well.
5	276	2M	Remained well.
2	276	2M	Remained well.
7	.....	.....	Lost, strayed or stolen after vaccination.
3	.....	.....	Died from castration.
1	.....	.....	Died soon after vaccination. See table I.
Litter 5 — Vaccinated May 9, 1924			
1	217	910I	Died 14th day. Hog cholera.
2	217	925I	Remained well.
4	217	735I	Reacted slightly.
5	217	975I	Died 13th day. Hog cholera.
6	217	975I	Died 14th day. Hog cholera.
3	224	2M	Remained well.
8	231	2M	Remained well.
7	235	2M	Remained well.
Litter 4 — Vaccinated May 15, 1924			
4	211	825I	Remained well.
7	211	725I	Remained well.
8	211	825I	Sick after injection. Died 3d day. Shock.
9	211	785I	Remained well.
3	218	2M	Remained well.
6	218	2M	Remained well.
1	270	2M	Remained well.
2	270	2M	Died 5th day. Rickets and hog cholera.
5	.....	.....	Died after operation for hernia.
Litter 2 — Vaccinated May 28, 1924			
4	198	750I	Remained well.
2	212	2M	Remained well.
5	216	2M	Remained well.
1	257	2M	Remained well.
3	257	2M	Remained well.
7	257	2M	Remained well.
6	.....	.....	Rickets. Killed.

TABLE II — *Continued*

Pig No.	Days from vaccination to trial injection	Virus given in trial injection—cc	Results of trial injection
		LITTER 7 — VACCINATED MAY 28, 1924	
2	212	2M	Remained well.
3	257	2M	Remained well.
1	.....	.....	Died after operation for hernia.
4	.....	.....	Died soon after vaccination. See table I.
5	.....	.....	Died soon after vaccination. See table I.
		LITTER 9 — VACCINATED JUNE 19, 1924	
1	235	2M	Died 6th day. Hog cholera.
2	235	2M	Remained well.
3	235	2M	Remained well.
4	235	2M	Remained well.
		LITTER 10 — VACCINATED JULY 3, 1924	
5	176	2M	Remained well.
1	221	2M	Remained well.
2	221	2M	Remained well.
3	221	2M	Remained well.
4	221	2M	Remained well.
		LITTER 12 — VACCINATED JULY 18, 1924	
1	161	2M	Remained well.
3	206	2M	Remained well.
2	.....	.....	7–30–24 taken out of this experiment and used for another purpose.
		LITTER 6 — VACCINATED JULY 21, 1924	
1	203	2M	Remained well.
2	203	2M	Remained well.
4	203	2M	Remained well.
3	.....	.....	Killed because of unthriftiness. Rickets. Ascariasis.

A total of 46 animals received the trial injection. Twelve taken from four litters were given as a trial injection 5 cc of virus per pound of live weight intravenously as is done in hyperimmunizing.

In litter No. 1, pig No. 3 died on the 9th day of hog cholera and No. 4 remained well.

In litter No. 5, pigs Nos. 1, 2, 4, 5 and 6 received for the trial injection 5 cc of virus per pound live weight. Nos. 1, 5 and 6 died of hog cholera. No. 2 remained well. No. 4 reacted but recovered. It is worthy of notice here that of the 3 other pigs of this litter that received 2 cc of the virus in the trial injection, all remained well in spite of the fact that they received their trial injection a longer time after vaccination.

In litter No. 4, pigs Nos. 4, 7, 8 and 9 received the 5 cc of virus per pound live weight intravenously. No. 8 showed signs of sick-

ness almost immediately and was dead the third day and showed no distinctive lesions on autopsy. This may have been hog cholera and it may have been shock as occasionally occurs in hyperimmunization. Three days is soon for death to occur from hog cholera as a lone cause. Of the four that received the trial injection of 2 cc of the virus intramuscularly, pig No. 2 died. This animal was in a bad condition with rickets when given the trial injection.

In litter No. 2, pig No. 4 was given the trial injection of 5 cc of the virus per pound live weight. This animal showed a rise in temperature the 9th and 10th days for some cause but otherwise remained normal and about two months later withstood hyperimmunization without showing signs of sickness.

SUMMARY.

Of the 12 animals that received the hyperimmunizing dose of virus of 5 cc per pound live weight intravenously, five died. Of the 34 that received the trial injection of 2 cc of virus intramuscularly, four died, and one of these had rickets badly when given the trial injection. Results in this experiment indicate a grade of immunity sufficiently high to withstand 2 cc of virus intramuscularly, which is easily sufficient to protect from natural exposure but not sufficiently high to protect from the enormous doses of virus used in the special process of hyperimmunization.

In 1922 and 1923, all animals that received a trial injection of 2 cc of virus intramuscularly remained well. A number of them were hyperimmunized after the trial injection without loss.

The different results in 1924 may have been due to one or more of the factors already mentioned. There are doubtless others that might cause them. About all that can be said with certainty is that a very important difference occurred and the fact that it did occur emphasizes the necessity of repeating these yearly experiments and doing further research on the factors which may be responsible for the variations observed.

The pigs in these experiments have not been as generally thrifty as pigs should be to be most profitable. The cause of the lack of thrift has not been determined.

REFERENCES

1. Report of the New York Veterinary College, 1922–1923, p. 204.
2. Report of the New York Veterinary College, 1923–1924, p. 87.

EXPERIMENTS WITH MIXTURES OF ANTI-HOG CHOLERA SERUM AND HOG CHOLERA VIRUS

J. W. BENNER
Veterinary Experiment Station

If serum and virus can be handled together where the simultaneous treatment is to be given it will save the time of bottling and administering the virus separately.

B. A. I. Circular 43,[1] issued February 12, 1904, contains information concerning the use of disease-producing blood and antitoxic blood and states that these products have been used successfully both separately and combined. Subsequent to the publication of Circular 43, McClintock, Boxmeyer and Siffer[2] attempted to use attenuated virus, also a combination of disease-producing blood and immune blood, but they were only partially successful.

In 1921, Duval and Couret[3] published experiments in which they had used a mixture of desiccated anti-hog cholera serum and desiccated hog cholera virus successfully. The action of a serum-virus mixture must depend on the potency of the products and the chemical and bacteriological properties of each, separately and when mixed. This gives rise to many possibilities and since very little is known about these properties, it seems that more experiments are justifiable.

A preliminary experiment was made by mixing 1 cc of stock virus with 50 cc of stock serum. Twenty-five cubic centimeters of the mixture were injected into the axillary space of a 50-pound susceptible shoat (No. 97) which was kept on hog cholera free ground (see Table I-A). Temperatures were taken daily for a week after the injection. No rise occurred. Seventy days later, 2 cc of potent virus were injected to test the immunity. The shoat proved to be immune.

TABLE I-A — MIXED SERUM AND VIRUS EXPERIMENT

Pig No.	Wt., lbs.	Virus (cc)	Serum (cc)	Method	Results
				MARCH 23, 1923	
97..	50	1/2	25	Serum mixed with virus 30 minutes and injected into axilla.	Remained well.
				AUGUST 15, 1923	
1..	30	2	10	Serum injected in one axilla, virus in the other.	Remained well.
2..	37	2	10	Serum injected into axilla, virus through same needle, same place.	Slight temperature reaction, but quickly recovered.
3..	33	2	10	Serum mixed with virus 30 minutes and injected into axilla.	Slight temperature reaction, but quickly recovered.
4..	68	2	.....	Virus injected into axilla..	Hog cholera.
5..	33	2	.....	Virus injected into axilla..	Hog cholera.

August 15, 1923, each of five shoats was given 2 cc of stock virus into the axillary space (see Table I-A). In one pig of this group, 10 cc of stock serum were given into the other axilla, constituting the regular simultaneous treatment. After the injection of virus another pig was given 10 cc of serum through the same needle before the needle was withdrawn. A third pig was given the 2 cc of stock virus which had been mixed with 10 cc of stock serum for 30 minutes. Pigs 4 and 5 were given the virus but no serum. The virus pigs showed a cholera temperature on the second day, were killed for virus the fourth day and showed lesions of acute cholera. Pigs 2 and 3 showed slight temperature reactions the sixth day and did not eat well but recovered quickly. February 11, 1924, pigs 1, 2 and 3 were given 2 cc of virus alone to test their immunity. Temperatures were taken daily for one week. No rises occurred. They remained well. The trial injections are not included in the tables.

TABLE I-B — MIXED SERUM AND VIRUS EXPERIMENT

Pig No.	Wt., lbs.	Virus (cc)	Serum (cc)	Method	Results
				AUGUST 22, 1923	
6..	33	2	7	Serum injected in one axilla, virus in the other.	Slight temperature reaction.
7..	33	2	7	Serum injected into axilla, virus through same needle, same place.	Hog cholera.
8..	31	2	7	Serum mixed with virus 30 minutes and injected into axilla.	Hog cholera.
				JANUARY 29, 1924	
101..	106	1	10	Serum injected in one axilla, virus in the other.	Slight temperature reaction.
102..	92	1	10	Serum injected into axilla, virus through same needle, same place.	Hog cholera.
103..	103	1	10	Serum mixed with virus 40 minutes and injected into axilla.	Hog cholera.
104..	82	1 3-mm loop	10	Mixed for 20 minutes and 2 cc injected into axilla	Hog cholera.
120..	106	2	.....	Virus injected into axilla.	Hog cholera.
121..	71	2	.....	Virus injected into axilla.	Hog cholera.

On August 22, 1923, I continued the experiment, using shoats of the same size with the same dose of virus (2 cc), but decreased the dose of serum used on August 15, from 10 cc to 7 cc. Pig 6 was given the serum and virus by the simultaneous method; pig 7 was given serum and virus through the same needle and pig 8

received the mixture (see table I-B). Pigs 7 and 8 sickened with cholera promptly while pig 6 remained well. Pig 6 survived this experiment, was also given 2 cc of virus on February 11, 1924, and remained well.

On January 29, 1924, I injected a group of larger shoats. They were of sufficient weight to give the same results with the 10-cc dose of serum that were observed with the 7-cc dose used in the group injected August 22. It will be seen in Table I-B that here again the animal which received the serum in one axillary space and the virus in the other withstood the vaccination while the animals receiving the same dose of serum and virus by the other two methods sickened with cholera. The animal that survived, No. 101, in the last group, was later exposed to hog cholera virus and found to be immune.

Changing the proportions of virus and serum in the three groups seemed to make no difference but it is indicated clearly that the method of injecting the virus at one place and the serum at another is more efficient than when they are mixed. This difference might not be noticed in practice where much larger doses are used but it was apparent in the small doses used in this experiment, where the breaking point of the serum was reached in order to test the methods.

The advantage of a serum-virus mixture would be greatly increased if the two substances could be placed together in the proper proportions by the manufacturer and used as a stock product. Here again the action of the mixture must depend on the potency of each product when mixed and the effect of time upon the potency of each, also the chemical and bacteriological properties of each, separately and when mixed. A mixture with an action analogous to the toxin-antitoxin mixture used successfully against diphtheria, or a product like a vaccine, which might permit the use of a smaller quantity of serum, would of course add favorably to the advantage in direct proportion to the decrease of serum permitted.

After the foregoing study of the efficiency of serum and virus used separately as compared with the fresh mixture of the substance, the following experiment was performed with the object of learning how long the mixture would produce a solid and permanent immunity when administered to pigs and shoats.

The dosage of the mixture used was about the same as the dosage of serum used in the regular simultaneous treatment. The serum and virus were mixed in the proportion of 20 cc of serum with 0.5 cc of virus. The virus was poured into the serum and the two agitated for about two minutes, or until it was thought the virus would be evenly distributed through the entire volume of serum. The mixture was then bottled and sealed as is done in the final preparation of serum that is to be sent out for field use. The serum used passed a good test begun on January 5, 1924. The virus used was bled on March 7, 1924. The mixing and bottling was done March 13, 1924. The mixture in all cases was administered by intra-axillary injections. Table II summarizes the data of the experiment.

TABLE 2—MIXED SERUM AND VIRUS EXPERIMENT

Pig No.	Wt. in lbs.	Date of injection of mixture	Dose of mixture, cc	Date of trial injection	Dose of virus, cc	Results of trial injection
139	75	4/14/24	40	7/11/24	2	Remained well.
140	75	4/14/24	40	7/11/24	2	Remained well.
142	170	5/15/24	70	7/11/24	2	Remained well.
143	175	5/15/24	75	7/11/24	2	Remained well.
144	125	6/13/24	60	11/13/24	2	Hog cholera. Recovered.
145	125	6/13/24	60	11/13/24	2	Hog cholera. Died.
146	125	7/11/24	60	11/13/24	2	Hog cholera. Killed.
147	125	7/11/24	60	11/13/24	2	Hog cholera. Killed.
149	150	8/16/24	75	.....	.....	.....
150	150	8/16/24	75	.....	.....	.....
151	175	9/17/24	80	.....	.....	.....
152	175	9/17/24	80	.....	.....	.....
153	100	11/13/24	70	12/ 5/24	2	Hog cholera. Killed.
154	100	11/13/24	70	12/ 5/24	2	Hog cholera. Killed.

It can be seen in Table II that two animals were vaccinated at about monthly intervals beginning with April 14, 32 days after the serum and virus were mixed. Nov. 13, 1924, 8 months after mixing and bottling, the last two animals were vaccinated. No animals were vaccinated in October.

Nos. 144 and 145, vaccinated June 13, 1924, when the serum-virus mixture was 3 months old, show that the mixture was beginning to lose its property of producing a high grade, lasting immunity. No. 144 showed a light form of cholera as a result of the trial injection but recovered; No. 145 a more severe form and died.

Nos. 146 and 147, vaccinated July 11, 4 months after the mixture was mixed and bottled, received a very low grade of immunity, if any, from the administration of the mixture. They contracted cholera in typical form after the trial injection.

Nov. 19, six days after Nos. 144, 145, 146 and 147 received the trial injection, 144 had a temperature of 104.2; 145—105.2; 146—107.2; and 147—107.2. These temperatures represent fairly well the severity of the disease as manifested by the clinical symptoms. Nos. 144 and 145 show in rather an exceptional way the mixture at a point between the time when it had a potency sufficient to immunize Nos. 142 and 143 and the time when it dropped so far below that potency that Nos. 146 and 147 received no noticeable protection against 2 cc of virus in the trial injection.

Nos. 153 and 154 were vaccinated with the mixture the same day the trial injection was administered to Nos. 144, 145, 146 and 147. This would probably not have been done had the results of the trial injection on the latter animals been known. However, the results of the trial injection on Nos. 153 and 154, given 22

days after the vaccination with the mixture, indicate that not only the potency of the virus had dropped but that possibly the potency of the serum had fallen below standard. Nos. 153 and 154 were purposely given extra large doses of the mixture, somewhat larger than would have been given in the regular serum alone treatment when as a rule a fair grade of immunity can be depended upon for 22 days.

Nos. 149, 150, 151 and 152 were not given the trial injection. The results of the trial injection on Nos. 144, 145, 146, 147, 153 and 154 show plainly enough what might have been expected if the trial injection had been given to Nos. 149, 150, 151 and 152.

CONCLUSION.

In these experiments, the immunity produced by injecting serum and virus in tissues far enough apart to insure separate absorption proved to be of somewhat higher grade than that produced by the use of the mixture made in vitro and in vivo. The one experiment described on a stock mixture of serum and virus indicates tendencies which may or may not become established when the work is repeated.

No product as a result of a neutralization of virus by serum, which would permit a decrease in dosage of the latter, was observed in either the fresh or stock mixtures. Each entity seemed to retain its properties and action regardless of the short period of contact as in the case of the fresh mixture, or the long period of contact as in the case of the stock mixture.

The stock mixture in the experiment described conferred a desirable grade of immunity in animals vaccinated as long as two months after the mixture was made.

REFERENCES

1. SALMON, D. E.; B. A. I. Cir. 43.
2. MCCLINTOCK, C. T., BOXMEYER, C. H. and SIFFER, J. J.; Jour. Inf. Dis., Mar. 1905, p. 351.
3. DUVAL, C. W. and COURET, M.; Jour. Med. Res., 1921.

INDEX

A

Abortion, 18, 54–58, 61–64, 84, 87–90, 100–133
Acanthosis nigrigans, 141–143
Accredited herd plan, 8
Actinomyces necrophorus, 167
Ambulatory clinic, 29–32
Anthrax, 45
Appropriations, 20, 21
Argus persicus, 223
Arteriosclerosis, 19, 163–172
Atherosclerosis, *see arteriosclerosis*
Autopsies, 17, 50, 51
Azoturia, 19, 197–199

B

Bacillus bronchi canis, 175
bronchisepticus, 175, 176, 184
caniculae, 174
coli, 87, 92, 93, 95, 96, 128
pestis, 176
pestiscaviae, 40
pyogenes, 54, 87, 95
Bacillary white diarrhea, 39, 40
Bacterins, 12, 15
Bacterium abortum, 18, 54–55, 57, 58, 60, 62, 79, 81–132
anatum, 40
coli, 175, 184
pullorum, 39
sanguinarium, 40
Bequests, 13
Bibliography, abortion, 110
arteriosclerosis, 170
azoturia, 199
Bacterium abortum, 110
blood, 199
bracken poisoning, 151
calf diseases, 22
distemper, 174–177, 188
fowl plague, 209, 214–216, 218, 224
faculty publications, 21, 22
genital diseases, 22
hog cholera, 21, 221, 235
poisoning, 53
poultry diseases, 21, 22
skin tuberculosis, 190, 192, 194
streptococcus, 161
tuberculosis, 190, 191, 194
urine, 199
Biologics, 49, 67; *see also hog cholera serum, tuberculin*
Blackleg, 45
Blood, 19
cattle, 200–205
horse, 197–199
Bracken poisoning, 18, 146–151
Brucella abortus, 8, 18, 44–46
Buildings, 11, 13, 20

C

Calf diseases, 17, 67–78; *see also Bacterium abortum*
Cat diseases, 36–38, 50, 51
mange, 137
rabies, 45
Cattle, abortion, 18, 100–133
blood, 19, 200–205
diseases, 30–35, 45, 46, 50, 51; *see also arteriosclerosis*
genital diseases, 52–66, 79–133
milk fever, 19, 200–205
mould, 127–133
poisoning, 146–152
rabies, 45
tuberculosis, 190–195
urine, 19, 200–205
Chlorides, *see urine*
Clinics, 11, 12, 16, 29–38
Colon organism, 54, 90, 91
Colostrum, 67–70
Conference for Veterinarians, 13–15
Creatinine, *see urine*
Curriculum, 8, 11–13, 15

D

Dermatocoptes cuniculi, 137
Diagnosis, 12, 15, 17, 44–49
Diarrhea, 39, 40
Distemper, 19, 173–189
Distocia, 59, 85
Dog, diseases, 18, 36–38, 50, 51, 134–146; *see also arteriosclerosis*
distemper, 173–189
rabies, 44, 45, 186
Dystocia, *see Distocia*
Dystrophia, 141–143

E

Entrance requirements, 7

F

Faculty, 8, 9
Feeding, 67–78
Fever, milk, 19, 200–205
Filaria equi, 164
Finances, 23, 24
Foot rot, 139
Fowl cholera, 40
plague, 20, 40, 41, 209–224
typhoid, 40
Foxes, 17, 45
Fracture, 206–208

G
Genital diseases, 17, 18, 29, 45, 46, 52–66, 79–99, 143–145; *see also abortion*
Gifts, 15
Glanders, 45
Graduate work, 12
Granuloma, 143–145

H
Hemorrhagic septicemia, 45
Hog cholera, 20, 225–235
  serum, 12, 46
Horse, azoturia, 19, 197–199
  blood, 19, 197–199
  diseases, 30–35, 50, 51; *see also arteriosclerosis*
  rabies, 45
  urine, 19, 197–199
Keratosis nigrigans, 141–143

I
Indigestion, 68, 69

K
Keel, 40
Keratosis nigrigans, 141–143

L
La grippe, 173
Lectures, 10
Library, 13
Live stock, 11
Lochia, 88
Lupus, 190, 191, 193

M
Mallein, 49
Mange, 134–139
Mastitis, 45, 109
Metabolism, 19
Metritis, 58, 66, 84, 87, 89, 90
Military training, 13
Milk, 69
  fever, 19, 200–205
Mould, 18, 85, 87, 90, 92, 94, 96, 127–133
Mycobacterium tuberculosis, 44

N
Needs of college, 20
Nervous distemper, 173–189 127–133

O
Open joint, 206–208

P
Paralysis, *see distemper*
Para-typhoid, 40
Pasteurella aviseptica, 41, 215
  canis, 174, 175
Peroxide, 154–162
Pneumococcus, 152, 153, 157
Pneumonia, 173
  calf, 67, 68
Poisoning, 18, 146–151
Poultry, 11
  diseases, 7, 8, 15, 17, 39–43; *see also fowl cholera, etc.*
Pseudomonas pyocyaneus, 184

R
Rabies, 44, 45, 186
Research, 16–20
Retained placenta, 58, 65, 84, 85, 87, 89
Ringworm, 138–141

S
Sarcoma, 143–145
Serum, *see hog cholera*
Sheep diseases, 30–35, 50, 51; *see also arteriosclerosis*
Skin tuberculosis, 19, 190–196
Small animal clinic, 36–38
Squirrel, rabies, 45
Staphylococcus, 87, 92, 94, 97, 184
  albus, 178
Sterility, 59, 84, 89, 90
Streptococcus, 18, 19, 54, 87, 90, 92–97, 152–162, 184
  alpha, 92
  hemolyticus, 87, 91
  mitior, 152
  pyogenes, 178
Strongylus larvae, 164
  vulgaris, 169, 170
Students, 10, 11
Sugar, *see urine*
Surgical clinic, 33–35
Swine diseases, 30–35, 50, 51; *see also arteriosclerosis, hog cholera*

T
Taenia pisiformis, 179
Tinea tonsurans, 139–141
Tuberculin, 12, 15, 19, 46, 49, 196
Tuberculosis, *see arteriosclerosis*
  fowl, 7, 41
  skin, 19, 190–196

U
Urine, 19, 197–205

V
Vaccines, 12, 15
Veterinary profession, 7, 8

W
White scours, 67, 68

X
X-ray, 20, 206–208

UNIVERSITY OF ILLINOIS-URBANA
3 0112 119663752